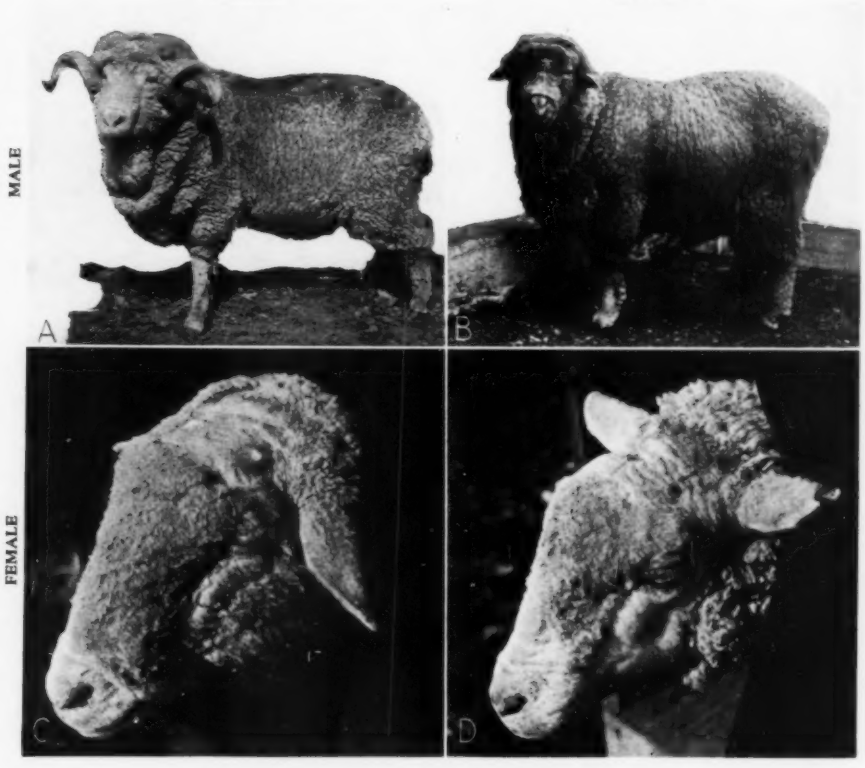
PEARL

The Journal of HEREDITY



SEX-DIFFERENCES IN RAMBOUILLET HORNS

(SEE PAGE 325)

Three Generations of Ear Pits

"Fused Follicles"—An Inherited Skin Defect

Breed Differences in Disease-Resistance in Horses

VOLUME 30 AUGUST, 1939 NUMBER 8

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

DAVID FAIRCHILD, President — Vice-President, W. E. CASTLE — Secretary, C. E. LEIGHTY
Treasurer, J. H. KEMPTON
Legal Advisor, STANTON C. PEELLE

EDITORIAL BOARD

Managing Editor, ROBERT C. COOK

M. DEMEREC — R. R. GRAVES — GEORGE M. DARROW
H. J. MULLER — PAUL POPENOE

Members of the Council are ex-officio members of the Executive Board

COUNCIL

W. E. CASTLE, Emeritus Professor of Genetics, Harvard University, and Research Associate of the Carnegie Institution of Washington.
LEON J. COLE, Professor of Genetics, University of Wisconsin.
O. F. COOK, Bionomist in Charge of Breeding Investigations, U. S. Department of Agriculture.
DAVID FAIRCHILD, Agricultural Explorer, U. S. Department of Agriculture.
HARRY V. HARLAN, Senior Agronomist in Charge of Barley Investigation, U. S. Department of Agriculture.
SAMUEL J. HOLMES, Professor of Zoology, University of California, Berkeley, California.
J. H. KEMPTON, Bureau of Plant Industry, U. S. Department of Agriculture.
W. V. LAMBERT, Geneticist, Animal Husbandry Division, U. S. Dept. of Agriculture, Washington, D. C.
C. E. LEIGHTY, Agronomist in Charge of Dry Land Agriculture, U. S. Department of Agriculture.
N. D. C. LEWIS, Director, New York State Psychiatric Institute and Hospital, New York City.
MARCUS M. RHOADS, Geneticist, Office of Cereal Crops and Diseases, U. S. Department of Agriculture, Washington, D. C.
SEWALL WRIGHT, Professor of Zoology, University of Chicago.

ADVISORY COMMITTEE

W. S. ANDERSON, Genetics Department, University of Kentucky, Lexington, Kentucky.
E. B. BABCOCK, Genetics Department, University of California, Berkeley, California.
E. A. BESSEY, Professor of Botany, Michigan State College, Lansing, Michigan.
A. F. BLAKESLEE, Plant Geneticist, Carnegie Institution, Cold Spring Harbor, New York.
W. E. BRYAN, Professor of Plant Breeding, University of Arizona, Tucson, Arizona.
C. B. DAVENPORT, Carnegie Institution of Washington, Cold Spring Harbor, L. I., New York.
RUSSELL W. DUCK, Livestock Editor, *The Rural New Yorker*, Syracuse, N. Y.
R. A. EMERSON, Professor of Plant Breeding, Cornell University, Ithaca, New York.
C. P. GILLETTE President, The Genetic Foundation, Fort Collins, Colorado.
C. M. GOETHE, Sacramento, California.
HARRISON R. HUNT, Professor of Zoology, Michigan State College, East Lansing.
H. S. JENNINGS, Professor of Zoology, Johns Hopkins University, Baltimore, Maryland.
D. F. JONES, Plant Breeder, Connecticut Agricultural Experiment Station, New Haven, Conn.
M. A. JULL, Senior Poultry Husbandman, U. S. Department of Agriculture, Washington, D. C.
F. B. LINFIELD, Director, Montana Experiment Station, Bozeman, Montana.
C. C. LITTLE, Director, Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine.
JAY L. LUSH, Professor in Animal Husbandry, Iowa State College, Ames, Iowa.
H. H. NEWMAN, Professor of Zoology, University of Chicago, Chicago, Ill.
T. S. PALMER, Expert in Game Conservation, U. S. Dept. of Agriculture, Washington, D. C.
JOHN H. PARKER, Professor of Agronomy, Kansas State College, Manhattan, Kansas.
JOHN W. SCOTT Professor of Zoology, University of Wyoming Laramie, Wyoming.
A. D. SHAMEL, Physiologist, U. S. Department of Agriculture, Riverside, California.
GEORGE H. SHULL, Professor of Biology, Princeton University, Princeton, New Jersey.
EDMUND W. SINNOTT, Professor of Botany, Barnard College, Columbia University, New York City. (Representative of A. G. A. on Council of Amer. Assoc. for the Advancement of Science.)
W. W. SMITH, Professor of Animal Husbandry, Purdue University, Lafayette, Indiana.
A. B. STOUT, Director of Laboratories, New York Botanical Gardens, New York City.
W. T. SWINGLE, Plant Physiologist in Charge of Breeding Investigations, U. S. Department of Agriculture, Washington, D. C.
W. H. TOMHAVE, American Aberdeen-Angus Breeders' Association, Chicago, Ill.
H. E. WALTER, Professor of Biology, Brown University, Providence, Rhode Island.
H. J. WEBBER, Prof. Subtropical Hort., Citrus Experiment Station, Riverside, California.
E. N. WENTWORTH, Armours' Bureau of Agr'l Research and Economics, Chicago, Illinois.
P. W. WHITING, Zoological Laboratory, University of Pennsylvania, Philadelphia, Pa.

The JOURNAL OF HEREDITY is published monthly by the AMERICAN GENETIC ASSOCIATION at 32nd Street and Elm Avenue, Baltimore, Md., and Victor Building, Washington, D. C. Address all communications regarding editorial matters to Room 308, Victor Building, Washington, D. C. Communications regarding the business management of The JOURNAL OF HEREDITY may be addressed to the publication office, 32nd Street and Elm Avenue, Baltimore, Md., or to the Victor Building, Washington, D. C. Entered as second-class matter February 24, 1923, at the postoffice at Baltimore, Maryland. Acceptance for mailing at the special rate of postage provided for in section 1103, Act of October 3, 1917, authorized December 20, 1918.

Contents copyrighted 1939 by the American Genetic Association. Unauthorized reproduction of material from THE JOURNAL not permitted. The title "Journal of Heredity" is registered as a trade-mark in the U. S. Patent Office.

$3.50 a Year. 35c the Copy. Prices of back numbers on application.

The

Journal of Heredity

Vol. XXX **AUGUST, 1939** **No. 8**

CONTENTS

THREE GENERATIONS OF EAR PITS
By D. D. Whitney 323

INHERITANCE OF HORNS IN SHEEP
By B. L. Warwick and P. B. Dunkle 325

A NEW INHERITED SKIN DEFECT IN MAN
By Milton H. Lehr 331

INHERITANCE OF LEAF AND FLOWER CHARACTERS IN TROPAEOLUM
By W. G. Whaley 335

INHERITED NON-BARRING OF THE FLIGHT FEATHERS IN TURKEYS
By V. S. Asmundson 343

DIFFERENCES IN INCIDENCE OF ENCEPHALOMYELITIS IN HORSES
By W. V. Lambert, S. R. Speelman and E. B. Osborn 349

Notes and Reviews

WHO DISCOVERED THE DOMINANT BLACK RABBIT? 324

BLOOD GROUPS A LA CARTE (Rev. of BOYD: *Blood Groups*)
By L. H. Snyder 329

MORE ADVICE FOR THE GROWING-UPS (Rev. of KELLIHER: *Life and Growth*)
By G. Haines 348

COMING

CALVIN B. BRIDGES

An appraisal of his personality and of his contributions to genetics.

THE QUINTS GROW UP

The Dionne quintuplets were quite different in weight and height when they were born, and yet they were considered to be identical. What has happened to them during five years of growth? Have their differences increased?

RECREATION AND EUGENICS

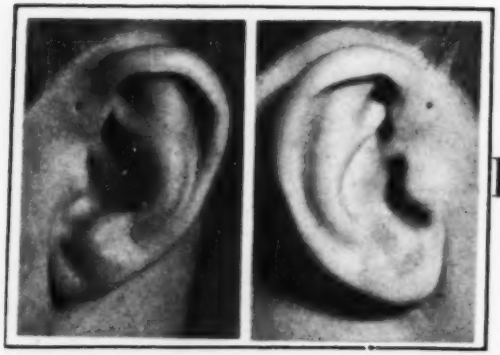
Public recreation has become a very large industry in recent years. What parts of the program have, or might have, eugenic significance?

A GENETIC INTERPRETATION OF EPIDEMICS

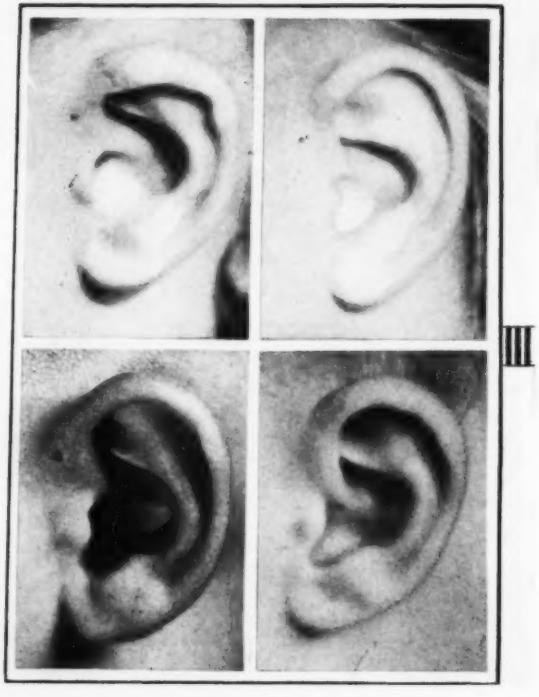
Do epidemics of disease have a genetic basis — hinging on the changing genetic immunity or resistance of the host population, and on the virulence of the pathogen?

$3.50 a Year **35c the Copy**

I



II



III

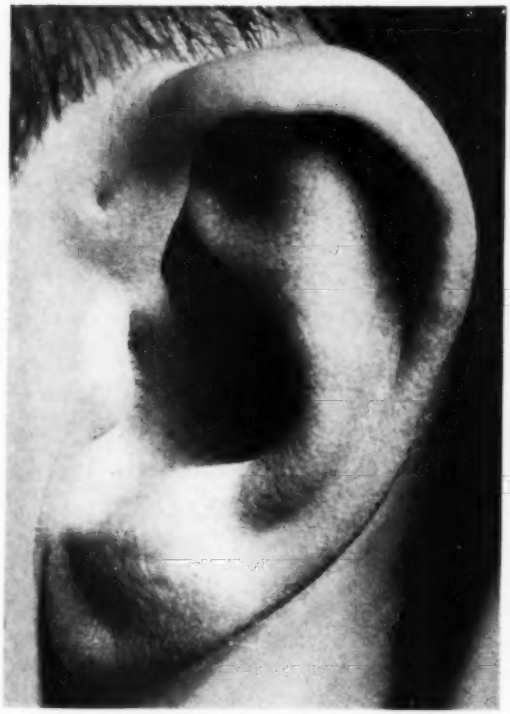
THREE GENERATIONS OF EAR PITS

Frontispiece

I. The left ear of the grandfather showing pit somewhat closed. II. The left and right ears of his son with pits well developed. III. Left ears of the four children of the son, three showing the pit. In inheritance the pit behaves as an irregular dominant, for in some cases parents who do not have the pit transmit it to their descendants. The pit is due to a failure to fuse of two embryonic tubercles which make up the human ear.

THREE GENERATIONS OF EAR PITS

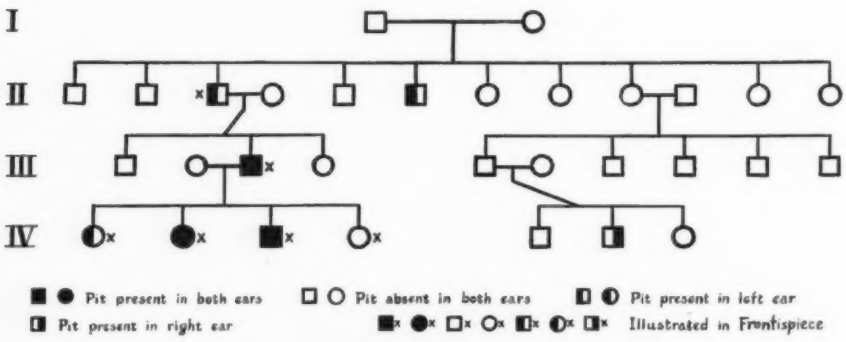
DAVID D. WHITNEY
Department of Zoology, University of Nebraska
Lincoln, Nebraska



DETAIL OF EAR PIT
Figure 1
A larger view of the left ear with pit of the son shown in the Frontispiece as II. The pits occur more frequently in the left ear than in the right

IN THE early stages of the human embryo the components of the external ear appear as several tubercles. Later these fuse to form the ear. In a few families tubercles 2 and 3 seem to fail to fuse completely, thus leaving a slight opening or pit at the proximal end of the upper part of the helix. Kindred has traced the inheritance of this pit in the left ear through four generations. In cases where one of the parents had the pit some of their children possessed it; in other cases, however, in which neither parent possessed it some of the children also had the pit—thus showing an irregularity in its appearance.

Ear pits in one or both ears in both



INHERITANCE OF PITS

Figure 2

Generation-I, parents without ear pits; generation-II, two sons (3 and 6) with a pit in left ear; generation-III, one son of II-3 with pit in left and right ear. In generation-IV one daughter (IV-1) had a pit in left ear; a daughter and a son had a pit in both ears; and one daughter had normal ears. A cousin (IV-6) had a pit in his right ear; neither his parents nor his grandparents had pits.

sexes have been found in another family, and as individuals of three generations are living, photographs have been made. Many of the affected individuals have the pit in the left ear and some in both ears but one individual has the pit in only the right ear. The grandfather, Figure 3 (II-3) having only the left ear pitted, has three children (III-1, 3, 4), one son and one daughter without pits and one son with pits in both ears. This son has four children (IV-1, 2, 3, 4), one daughter with a left pit, one daughter and one son with pits in both ears and one daughter free from pits.

In this particular family the presence of ear pits behaves as a simple dominant. A relative without pits, however, in another branch of this family, Figure 2 (III-5), has a left-handed son (IV-6) with a pit in only his right ear. This skipping of several generations may indicate that there are modifying genes involved or the possibility that the gene for pits mutates frequently in this family.

These observations corroborate the findings of Kindred and in addition show that the pit may occur very frequently in both ears and in both sexes.

Literature Cited

KINDRED, JAMES E. Inheritance of a pit in the skin of the left ear. *Jour. Heredity* XII: 366-367, Fig. 1. 1921.

Who Discovered the Dominant Black Rabbit?

TO THE EDITOR:

My friend, Richardson, an authority in his own right on the genetics of rabbits, is quite correct in saying (under the above heading, in the May number of the JOURNAL) that Punnett, not I, discovered dominant black. I am grateful to him for pointing out the error. It probably arose from the fact that in my little book summarizing knowledge of rabbit genetics as it then existed, I did not (for the sake of brevity) go into the history of the individual discoveries. This has been done adequately by Nachtsheim in a subsequent publication.

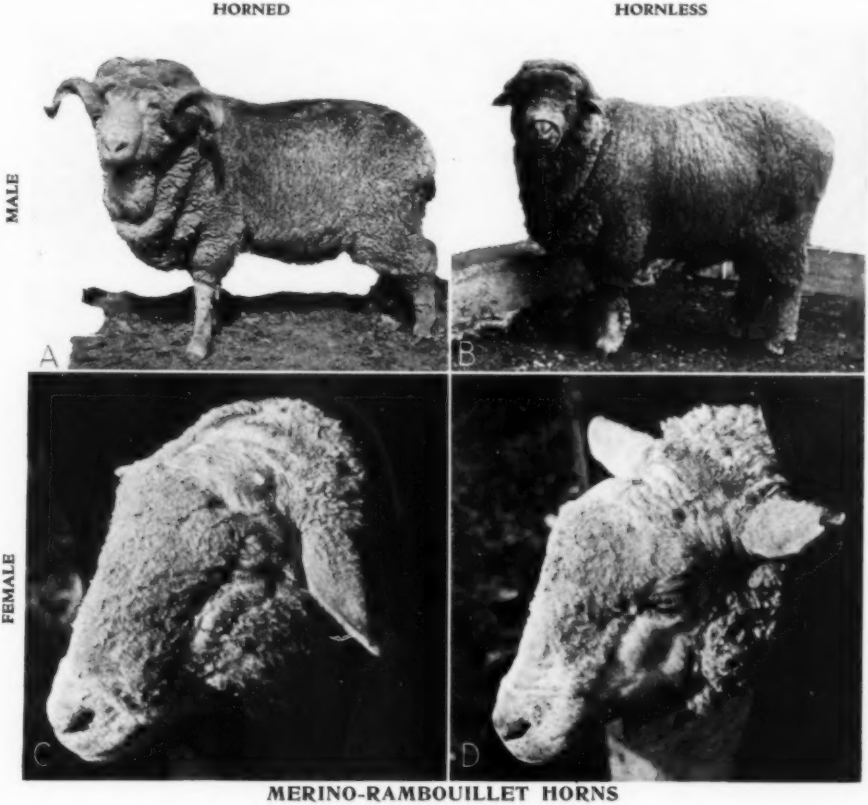
W. E. CASTLE

University of California

INHERITANCE OF HORNS IN SHEEP

Triple Alleles in a Dorset-Rambouillet Cross

B. L. Warwick and P. B. Dunkle*
Agricultural Experiment Station, A. and M. College of Texas



MERINO-RAMBOUILLET HORNS

Figure 3

A—Merino ram showing typical horns of the Merino-Rambouillet type. This is the recessive of a series of triple alleles, designated *hh*. A Rambouillet ram homozygous for hornlessness (*HH*) is shown at *B*. In females of the Merino group the horns are almost completely suppressed. *C* shows a "horned" Rambouillet ewe (*hh*), with wool clipped to show the knobs, which sometimes break through the skin to form scurs. The hornless ewes (*HH* or *Hh*) have actual depressions instead of either knobs or scurs, as shown at *D*.

RAMS of many breeds of domestic sheep have horns. The horns of some are relatively small, while others are very large and approach in size those of the Argali and Rocky Mountain Bighorn wild species. Rams' heads of these wild species have been among the most coveted trophies

*The authors wish to acknowledge the cooperation of Mr. Dantes Reiley, Sonora, Texas, who raised part of the lambs in this study.

THE DORSET HORN

Figure 4

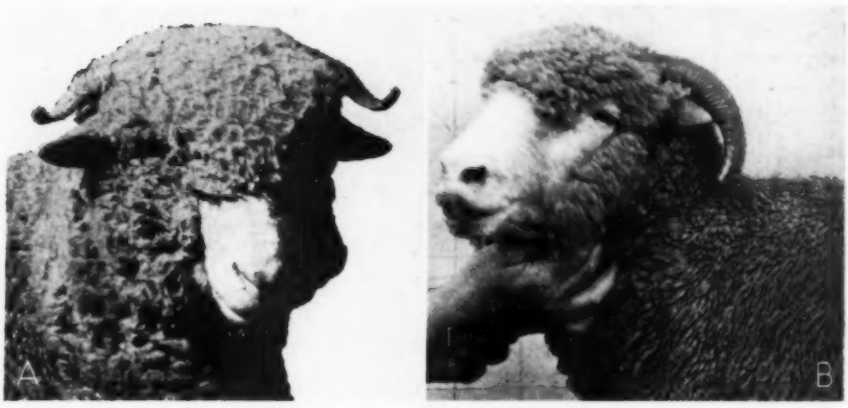
In the Dorset Horn breed both sexes are horned. The ram has a much larger horn than the ewe. The genotype of these sheep is *H'H'*. Crosses of Dorsets and Merinos are shown in Figure 5. Photographs courtesy of the Continental Dorset Club.

sought by big game hunters ever since the time of Marco Polo. One of the domestic breeds in which the rams have very large horns is the Dorset Horn which originated in Dorsetshire, England. In this breed the females also have horns, resembling in this respect most of the wild species. The horns of females, however, when present in any breed or species, are always much smaller than those of the males.

The Merino group (including the Rambouillet) is the most widely distributed and the most numerous of the breeds or types of domestic sheep. In most strains of this type, the males have horns while the females are hornless. These females have knobs which consist of protrusions from the skull resembling horn cores, except that they are usually less than an inch in height and are covered with skin. In some cases these have small horn-like coverings designated as scurs. These scurs are usually easily distinguishable from the horns of females of such breeds as the Dorset Horn because the scurs are small and irregular in shape. The Mouflon, which is one of the types of wild species represented in the ancestry of domestic sheep, also has horns on the males and knobs or scurs on the female.

Many breeds of domestic sheep are hornless in both sexes. Representatives of these breeds are the Shropshire, Southdown, Cotswold, and Suffolk. These animals not only lack horns, but have depressions in the skull instead of horn cores such as found protruding from the skulls of the horned breeds. These depressions may be easily identified on the living animal by examination. There are also strains of Merinos and Rambouillets in which most of the rams are hornless, and most of the females have depressions instead of knobs or scurs. These will be referred to in a later portion of this paper.

It is not surprising that when Mendelism was rediscovered, a study of the inheritance of horns should be one of the first such studies with mammals. Wood[7,8] reported in 1905 and 1909 the results of crossing the Dorset Horn and Suffolk breeds. On the basis of his carefully conducted experiments he interpreted the inheritance of horns as dominant in the male and recessive in the female. This early work established



DORSET × MERINO HYBRIDS

Figure 5

Hybrids show the interaction of the Dorset and Merino horn allels in crosses. *A* shows a backcross ewe, whose mother was a Rambouillet and whose sire was a hybrid Dorset × Rambouillet. Her genotype is *H'h*. *B* is an F_1 Merino × Dorset ewe (genotype *H'h*) showing typical Dorset horns. This cross is produced commercially in Pennsylvania and Ohio for the raising of hot-house lambs. Photo *B* courtesy Professor W. L. Henning of Pennsylvania State College.

the main facts as to what takes place in the F_1 and F_2 of this cross. However, there have been differences of opinion as to the interpretation. His statement that horns are dominant in the male and recessive in the female is predicated on the occurrence of horns on the F_1 males and on the males of the same genotype in F_2, while females of the same genotype are hornless. Wood recognized that his interpretation of dominance in the male was based on meager evidence as he stated, ". . . it must be remembered that only one F_1 ram was kept entire until adult and his horns never attained the size or shape of those of the parent Dorset . . ." Arkell[1] also reported horned F_1 rams when he crossed the Dorset Horn with hornless breeds, but the horns were smaller than those of the parent Dorset. Accordingly, the horns of F_1 rams approach the intermediate. Arkell proposed another hypothesis, involving a completely sex-linked inhibitor, which has since been discarded. Wood's interpretation has been generally accepted. However, when the F_1 phenotype approximates the intermediate, as in this case it becomes a question of individual preference, as to which parental type is considered dominant. Wood might well have considered hornlessness as dominant in both sexes, but with dominance incomplete in the male. We now have data which indicate that this is the valid interpretation.

The F_1 ewes reported by Wood all had depressions such as are typical of the strictly hornless breeds. After they became two or three years old, however, they threw up small scurs about one-half inch high. These scurs formed at maturity in the depressions are not to be confused with the typical knobs on the skulls of Merino and Mouflon ewes.

In addition to the crosses of hornless breeds with the Dorset Horn reported by Arkell[1] in 1912, his report included crosses of hornless breeds with Merinos and Rambouillets. He considered horns of the Merinos as dominant in the male and recessive in the female, but with less "potency" than in the Dorset. This was to account for the knobs or scurs of the Merino females instead of horns in the females as in the Dorset breed.

He also included a sex-linked inhibitor.

In 1931 we[3] presented evidence that the horns of Rambouillet males and the knobs of the females were due to one principal pair of recessive genes. This was based on the crossing of purebred Rambouillet rams of a hornless strain with horned-bred Rambouillets. One of the rams proved to be homozygous for hornlessness. About half of his F_1 progeny had scurs, but none had full size horns. Since then other homozygous rams have been identified by breeding tests[5,6]. Also, segregation has been within normal expectations. The occurrence of some F_1 and other heterozygous rams which are completely free from any trace of horn or scur is significant. We are inclined toward the view that scurs may be due to additional minor genes, but our tests are not yet complete. The mono-factoral basis for the inheritance of hornlessness and horned in these stocks has been verified and will be reported in detail in another paper. Apparently the reason Arkell missed the correct interpretation was because of the extremely small number of F_1 males studied and to his considering even the merest trace of scur as a horn.

Merino ewes are frequently crossed with Dorset Horn rams in Pennsylvania and Ohio for commercial purposes.[2] The F_1 females are especially liked for the raising of so-called hot-house lambs. When the parental Merino ewes have knobs as is usual, the F_1 ewes all have horns rather than knobs like their mothers. Many such F_1 ewes have been examined by the senior author. When homozygous hornless Rambouillet rams, such as those we identified by breeding tests or rams of pure hornless breeds as the Southdown or Shropshire are crossed with Merino or Rambouillet ewes, the F_1 female progeny are invariably hornless (with depressions).

Accordingly, the two breeds or types, Dorset which is horned in both sexes, and Merino (which includes Rambouillet) which has horns on the male but the corresponding females of which have knobs, when respectively crossed with the pure hornless type, produce hornless (depression) female progeny in every case. When crossed with each other, they produce horned females like the Dorset parental type. These facts suggested the hypothesis that the genes for hornlessness, Dorset horns, and Merino horns constitute an allelic series. In order to test this, we bred an F_1 Dorset $\times$ Rambouillet ram to Rambouillet and Merino ewes which had knobs. If the horns of these two breeds or types, Dorset and Merino (including Rambouillet), instead of being allelic were due to different pairs of genes, we would expect the Dorset to carry the genes for hornlessness which would be alleles of the genes for horns in the Merino and would expect the Merino to carry the genes for hornlessness which would be alleles of those for horns in the Dorset. It would then be logical to expect the F_1 females to be hornless rather than horned. Then the back-cross mentioned above should yield horned, knobbed and hornless (depression) females in a 1:2:1 ratio. If the genes for hornlessness (H), Dorset horns (H') and Merino (or Rambouillet) horns (h) are allelic, as postulated, the back-cross would yield female progeny of only two phenotypes, horned and knobbed in 1:1 ratio. This was found to be the case. Forty-nine lambs, twenty-two of them females were raised. Eight were horned, 14 were knobbed. This is within the normal sampling range for 1:1 ratio[4]. None had depressions. All of the ram lambs were horned. Ewe lambs with depressions would have been expected on the basis of two pairs of genes. If it should be assumed that the 25 per cent which would have had depressions were missed by chance, the probability of this would be .0018 or only 18 chances in ten thousand[4]. The above data are considered conclusive.

Summary and Conclusions

To test whether the genes for hornlessness (H), Dorset horns (H') and Merino (and Rambouillet) horns (h) were allelic, one F_1 Dorset $\times$ Ram-

bouillet ram was back-crossed to Merino and Rambouillet ewes with knobs. Eight of the females progeny had horns like F_1 Dorset $\times$ Merino ewes while 14 had knobs like the Merino and Rambouillet ewes. None had depressions such as would be expected on the basis of two pairs of genes. The genes H, H', and h are considered to be multiple alleles.

Literature Cited

1. Arkell, T. R. Some data on the inheritance of horns in sheep. New Hampshire Exp. Sta. Bul. No. 160. 1912.

2. Henning, W. L., P. T. Ziegler, and P. C. MacKenzie. Producing early lambs. Pennsylvania Agric. Exp. Sta. Bul. No. 255. 1930.

3. Warwick, B. L. The inheritance of horns in fine-wool sheep. *Anat. Record.* 51 (supl.) :116. 1931.

4. ————. Probability Tables for Mendelian ratios with small numbers. Texas Exp. Sta. Bul. No. 463. 1932.

5. ————, J. M. Jones, W. H. Dameron, and P. B. Dunkle. Inheritance of polled character in fine-wool sheep. Texas Agric. Exp. Sta. annual reports for 1931-1937.

6. ————. Polled Rambouillet breeding. *Proc. Am. Soc. An. Prod.* 1933.

7. Wood, T. B. Note on the inheritance of horns and face-colour in sheep. *Jour. Agric. Sci.* 1 :364-365. 1905.

8. ————. The inheritance of horns and face-colour in sheep. *Jour. Agric. Sci.* 3 :145-154. 1909.

BLOOD GROUPS A LA CARTE

THERE have been several excellent summaries of present knowledge regarding blood grouping, both text books and articles. The present volume* might therefore seem superfluous, were it not for the fact that it differs from all preceding works in one important way. The material is presented almost entirely in tabular form, with a minimum of textual matter. Thus the important factual information is readily available in compact concise form. The presentation is in three parts. The Introduction, with six tables, sets forth the plan of the work and outlines the various phases of the blood group problem. The General Part is concerned with the aspects of blood grouping other than anthropological. The Anthropological Part is devoted entirely to racial data.

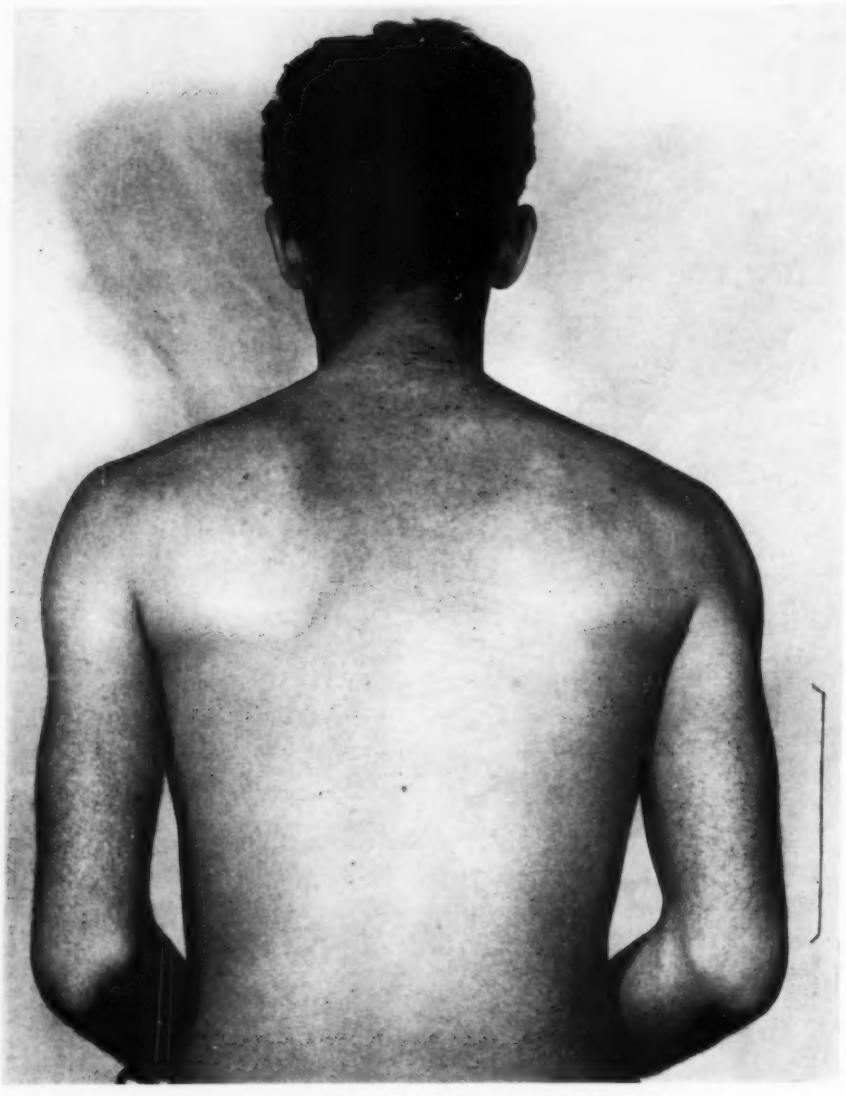
Sixty-four tables make up the General Part, ranging through such topics as the serologic basis for the groups, their inheritance, their occurrence in various tissues, organs and species, the subgroups, medico-legal applications, technic of testing stains, technic of studying mummies, and the relationship of the groups to disease. In the various topics are included the M-N type as well as the A-B groups.

The Anthropological Part opens with a page of discussion. Then follow two maps, showing the gene frequencies of A and B, respectively, with contour lines drawn through points of equal gene frequency. A table is presented giving standard deviations of blood group frequencies. Three nomograms simplify the determination of gene frequencies and their standard deviations. The bulk of the presentation is given over to two tables, of the A-B groups and the M-N types, respectively, in which are listed geographically the results of studies on the various peoples of the world. These tables have been very carefully collected and put together, and should be of great value to geneticists and anthropologists. Approximately 1,00 separate listings are given in the A-B tests, and about 130 in the table of M-N. Nearly 400 references are cited in the bibliography.

The advantages of tabular presentation of otherwise bulky material are readily apparent. The disadvantages, while not so apparent, are nevertheless real. There is no opportunity for discussion, no possibility of critical evaluation, and no room for speculation. Much faith must be placed in the author in his choosing and selecting of material to be presented. In this case such faith seems wholly justified.

Laurence H. Snyder
The Ohio State University

*Boyd, W. C. Blood Groups. *Tabulae Biologicae* vol. XVII, par 2. 128 pp. 73 tables, 13 figures, 3 maps. The Hague (W. Junk). 1939.



LOCATION OF SPIRALLED HAIR FOLLICLES

Figure 6

This inherited abnormality of the skin (Keratosis Follicularis Spiralis) is manifested chiefly on the upper arms, and on the legs, thighs, and buttocks, affecting only the extensor surfaces. The bracket indicates the region on the arms in which the follicles are abnormal, producing a bumpy effect. The skin of the back and shoulders is normal. The subject shown here is III-23 in Figure 7, Family *A*.

A NEW INHERITED SKIN DEFECT IN MAN

Pedigrees of Two Families With a Sex-Influenced Hereditary Keratosis Follicularis Spiralis

MILTON H. LEHR

Carnegie Institution of Washington, Cold Spring Harbor, N. Y.
Virginia Polytechnic Institute, Blacksburg, Va.

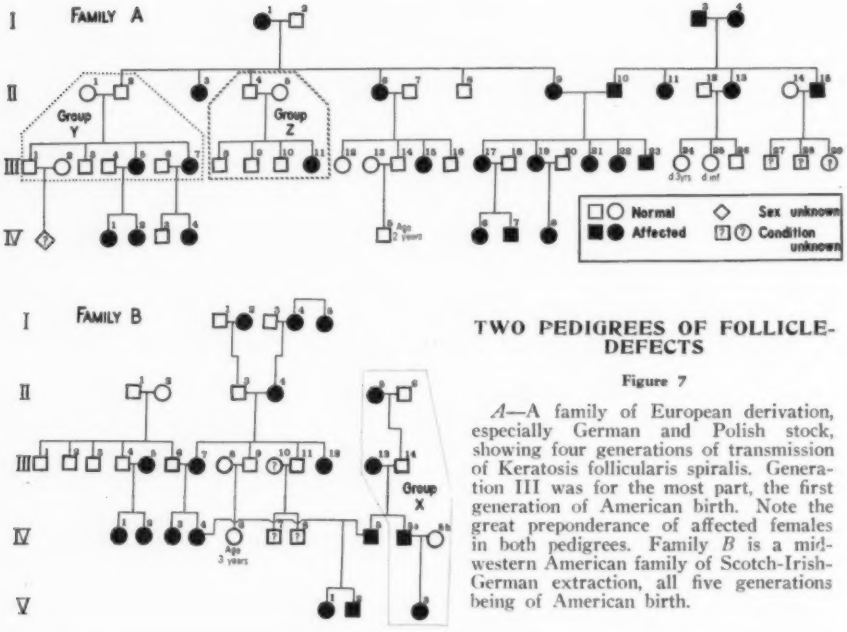
HEREDITARY keratosis (variously called keratoma, tylosis, lichen, etc.) has been described and recorded in many genetic sources. H. W. Siemans[7] worked out the inheritance of several keratosic conditions and found that there are a number of distinct forms ranging in severity of symptoms from keratosis in its most extreme form (K. follicularis) to normal skin. Medical studies by Schamberg,[6] Bulkley,[2] and Radcliffe-Crocker[5] record many of these transitional forms all under the heading of *hypertrophies of epithelium*, seemingly similar in nature, causes, treatment, and perhaps inheritance. In general Siemans says that the probability for polymeric causation does exist. However, morphological and genetical investigations of many and varied skin ailments, indicate that in the types which are hereditary the genetic basis has usually proven to be rather simple—dominance or recessiveness with occasional sex-linkage. (See Table I.)

Although many similar conditions have been reported, Keratosis follicularis spiralis* has not been described medically or genetically. It makes its appearance on the extensor surfaces of the arms and legs as a hypertrophic affection characterized by pinhead-sized epidermal accumulations at the mouths of the hair follicles. The lesions consist of closely aggregated, pinhead-sized, conical elevations corresponding to the orifices of the hair follicles. The lesions are whitish in color and are made up of epidermal cells and sebum. As a result of the cornification of the follicles, the growing hairs are unable to penetrate the surface of the skin. In spite of the adverse conditions for growth, the hairs do not perish but spiral about in the follicle while continuing vegetative processes. The skin is rough and dry and has the appearance of "goose-flesh." (See Figure 6.)

The ailment differs from similar conditions (Lenz's K. follicularis, Lichen pilaris, etc.) in that the hairs do not perish; the disease, beginning at about five years of age and climaxing at adolescence, does not regress but remains the same throughout adulthood; horny nodules are formed; white scars do not replace the cornified areas; there are no pit-like depressions; it appears *only* on the extensor surfaces of the arms, legs, and buttocks; and the lesions are usually whitish or flesh-colored, never brown or black.

Lenz[4] describes a K. follicularis (lichen pilaris) in which the hair follicles undergo cornification on the extensor surfaces of the arms and legs. Horny nodules are formed and are later replaced by white scars. Lenz calls this condition a dominant manifestation. Trimble[10] reported the same condition but described it in a considerably different manner. He states that the malady appeared about the scalp, forehead, temples, legs, back, and chest.

*For their generous medical assistance I wish to thank Drs. B. Fabricant, H. C. Comora, and C. L. Littauer, all of New York City. The terminology employed in determining the name for this condition was suggested by them as follows: keratosis—relating to a disease of the epidermis with circumscribed overgrowths of the horny layer; follicularis—relating to the follicles (as in the cornified condition of the hair follicles in this case); spiralis—relating to the spiraled growth of the encased hairs within the hardened follicle.



TWO PEDIGREES OF FOLLICLE-DEFECTS

Figure 7

A—A family of European derivation, especially German and Polish stock, showing four generations of transmission of Keratosis follicularis spiralis. Generation III was for the most part, the first generation of American birth. Note the great preponderance of affected females in both pedigrees. Family *B* is a mid-western American family of Scotch-Irish-German extraction, all five generations being of American birth.

The lesions formed were light brown in color varying in size from that of a pin-head to the head of a small match. Many lesions showed a central pit-like depression. Here too the disease was a simple dominant. Goldenberg[3] described a condition quite similar to Lenz's K. follicularis except that it appeared only about the neck and head. Again the trait was dominant. An overlapping ailment differing in degree and type of inheritance was reported by Siemans.[4] Keratosis follicularis spinulosa decalvans, small spiny and horny prominences on the arms and face with inflammation of the eyelids, was described as being "intermediate" sex-linked.

Further investigation of other than keratosis skin conditions showed that a rather regular scheme of inheritance manifests itself throughout dermatological inheritance. In keeping with the keratosic reports mentioned here, as well as the cross-sectional matter contained in Table 1, we are in a position to assume that, in general, hereditary skin diseases usually follow a dominant-recessive scheme, in some cases strictly sex-linked, and in some cases showing an irregular manifestation. The symbols involved in the discussion of this trait can be best represented as follows: *F*—to represent a gene for Keratosis follicularis spiralis. This is sex-influenced so that it is dominant in women and recessive in men. *f*—to represent a gene for normal skin.

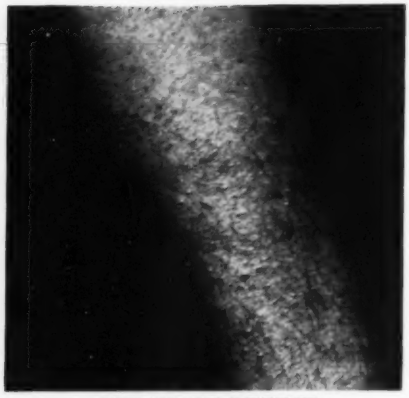
According to the data found in these pedigrees, transmission of the trait seems to be directly influenced by the sex of the individual. The fact that so many more females than males show the trait indicates that factors other than chance are involved in the scheme of inheritance. In a truly sex-influenced trait (one in which the condition is dominant in one sex and recessive in the other) we would have the following genotypes: *FF*, an affected female or an affected male; *Ff*, an affected female or a normal male; *ff*, normal in either sex. There would therefore be three times as many females showing the trait as males. The ratio of

TABLE I. Abstracted from Lenz's review[4] of morbific hereditary factors.

Investigator	*Description of Disease*	*Type of Inheritance*
Davenport & Preiser	Neurofibromata—naevoid affection originating in the cutaneous nerves.	Dominant.
Siemans & Henle	Telangiectases—localized dilation of capillaries in the skin.	Dominant.
Siemans	Naevi pigmentosi—white patching in the skin.	Dominant.
Hammer & Meirowsky	Ephelides—freckles.	Dominant.
Siemans	Ephelides—freckles.	Cooperative factors.
Fasold	Xanthomatosis—dark yellow raised patches on the skin—deposits of cholestrin.	Dominant (but does not manifest itself in all persons who carry it).
Kohn & Siemans	Xeroderma pigmentosum—normal skin at birth followed by freckling and cancerous changes with death occurring before 20 years of age.	Recessive.
da Costa	Bullous disorder attended by severe general disturbances.	Recessive and sex-linked.
Siemans	Anidrosis—complete failure of sweat glands to function—poor development of teeth and hair.	Recessive and sex-linked.
Lenz	Hyperidosis—excessive secretion of sweat.	Dominant.
Leven	Ichthyosis vulgaris—skin covered with dense scales and horny flakes.	Dominant.
Lenz	Anonychia—absence of nails; Leuconychia—white patches in nails.	Dominant.
Snyder*	Epidermolysis bullosa—slight friction or abrasion of the skin results in formation of blebs or blisters. *Not included in Lenz's review.	Dominant.

TABLE II. All possible unions in sex-influenced inheritance with representative extracts from pedigrees of Families *A* and *B*.

♂	×	♀	*Theoretical Offspring*	*Pedigree Abstract*
FF	×	*FF*	All affected.	Fam. *A*; Gen. I; 3, 4 producing all affected; Gen. II; 10, 11, 13, 15.
FF	×	*Ff*	½ ♂ affected; all females affected.	Probably Fam. *A*; Gen. II; 9 and 10 producing all affected females and one theoretically possible male.
FF	×	*ff*	All females affected; no males affected.	Fam. *B*; Gen. IV; 5a, 5b producing, so far, one affected female.
Ff	×	*FF*	All females affected; ½ males affected.	Fam. *A*; Gen. III; 17, 18 producing one affected ♀ and, so far, one affected male.
Ff	×	*Ff*	♂ = 1 aff.; 3 normal; ♀ = 3 aff.; 1 normal.	Several possibilities but no suitable extract.
Ff	×	*ff*	½ females affected; no males affected.	Fam. *A*; Gen. II; 4 and 5 producing three normal males and one affected female.
ff	×	*FF*	All females affected; no males affected.	Fam. *B*; Gen. II; 3 and 4 producing two affected females and two normal males.
ff	×	*Ff*	½ females affected; no males affected.	Fam. *A*; Gen. III; 6 and 7 producing one normal male and one affected female. Fam. *A*; Gen. II; 6 and 7 producing two normal males, one normal female, and one affected female.
ff	×	*ff*	All normal.	Fam. *B*; Gen. II; 1 and 2 producing five normal males. (No female offspring at all.)



DETAIL OF DEFECT

Figure 8

Close-up of upper arm of III-23, showing the bumpy skin caused by the follicle defect.

affected females to normal females would also be 3:1, because of the two trait-carrying genotypes to the one normal. Consequently our pedigrees showing 37 out of 44 females affected is in accordance with the previous assumption.

In a union where only the male parent was affected, we would have the following cross: FF ♂ × ff ♀ = Ff (all females affected; all males normal). From such a cross we would conclude that the trait would always appear in the daughters of an affected father. In the pedigree of Family *B* (Group *X*, Figure 7) this type of cross is illustrated. The marriage IV-5a and 5b is FF ♂ × ff ♀, and resulted in the heterozygous affected female, and a prediction for a normal male in conformity with the theory.

Where neither parent showed the trait but the father carried the gene (Ff ♂ × ff ♀), we should expect half the daughters to be affected. Three types of offspring produced would be expected: Ff "carrier" males, Ff affected females, and ff normal males and females. In Family *A* (Group *Y*) II-1 and 2 represent the union of a carrier male with a normal female. Daughters III-5 and 7 represent the heterozygous females, and III-1 and 3 represent the normal males, who theoretically could not be affected. The sex-influenced nature of this ailment is also shown in the children of II-4 (Family *A*, Group *Z*), where again all males are normal (one-half of them carriers) and the female expresses the trait. Table II shows in brief form every type of inheritance involved in sex-influenced inheritance in the families studied.

Summary

Keratosis in its varied forms has been explained genetically as being a dominant-recessive trait. Bateson[1] has said in reference to his work with several pedigrees of tylosis, "In view of the complete escape of the offspring of the unaffected except in one family tree the fact that tylosis (called also keratosis) almost always behaves as a simple dominant may be regarded as established." Lenz[4] states that all hereditary keratosis, keratomae, and tylosis are regularly dominant. In spite of the varied reports mentioned before the definiteness of Lenz and Bateson's statements, new, complete data of a previously unreported keratosis, that of K. follicularis spiralis, indicates that possibilities for sex-influence exist in hereditary skin conditions.

Literature Cited

1. Bateson, W. Mendel's Principles of Heredity. Cambridge University Press. pp. 219. 1930.

2. Bulkley, I. Duncan. Manual of Diseases of the Skin. G. P. Putnam's Sons. New York and London. pp. 244. 1903.

3. Goldenberg. Keratosis Follicularis Capitis (Two Cases, Father and Daughter). *Journal of Cutaneous Diseases*, Vol. 29. pp. 553.

4. Lenz, Baur, Fischer. Human Heredity. The Macmillan Company, New York. pp. 268-285. 1931.

5. Radcliffe-Crocker, H. Diseases of the Skin. P. Blakiston's Son and Company, Philadelphia. pp. 452-454. 1905.

6. Schamberg, Jay Frank and Carroll S. Wright. A Compendium of Diseases of the Skin. P. Blakiston's Son and Company, Philadelphia. pp. 200-201. 1934.

7. Siemans, H. W. Ueber einen in der menschlichen Pathologie noch nicht beobachten Vererbungsmodus: dominant-geschlechtsgebundene Vererbung. *Archiv fur Rassen und Gesellschaftsbiologie*, Vol. XVII, No. 1. 1925.

8. Snyder, Lawrence H. Principles of Heredity. New York. D. C. Heath and Company. 1935.

9. Trimble, William B. Observations on Keratosis Follicularis with Report of Five Cases in the Same Family. *Jour. of the Amer. Med. Assoc.* Vol. 59. pp. 604-607.

INHERITANCE OF LEAF AND FLOWER CHARACTERS IN THE NASTURTIUM

W. G. WHALEY*

THE general purpose of this investigation has been to study thoroughly the inheritance of several contrasting characters with the hope that one or more might prove amenable to a detailed developmental study after the genetic background was known. For this purpose a large number of characters was observed. Some of these have proven to be not clearly enough defined, and some of them too complex for analysis in the small populations which it has been possible to produce with only limited space available, and each cross producing only one or two seeds. Several of the simpler ones are presented here.

The greater part of the data have been derived from the observation of first and second generation and backcross hybrids between two species of Tropaeolum; Golden Gleam, a horticultural variety of the species *majus*, and *T. peltophorum fimbriatum*, a species bearing lobed leaves and small open flowers with dentate petals. Material from a few other crosses made by the writer is considered in some instances, and in one or two cases some hitherto unpublished data collected by Shaw[11] are added.

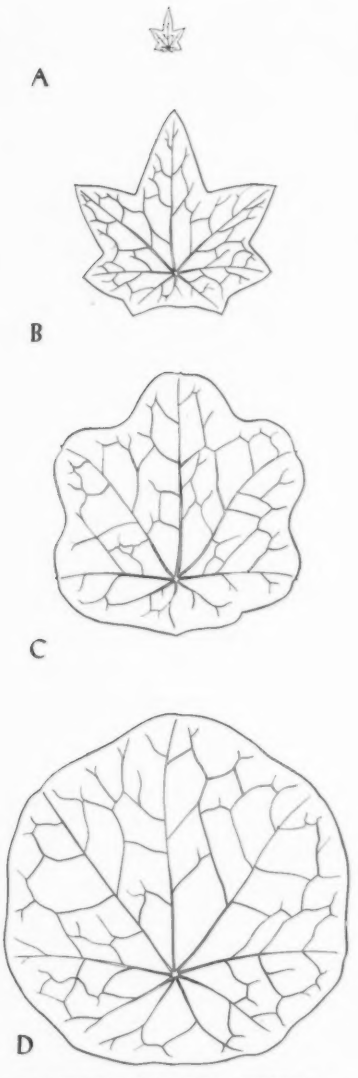
The Leaf

Shape.—The leaves of the members of the genus Tropaeolum are alternate, and either peltate or digitately lobed or dissected. The present study was confined to the variations occurring within the peltate leaved group. *Tropaeolum majus* var. *Golden Gleam* bears leaves which are broadly orbicular in shape (Figure 9*D*). The leaves of *T. peltophorum fimbriatum* conform to the same basic pattern but are characterized by three distinct lobes, and two less pronounced ones nearer the base of the leaf (Figure 9*C*). The hybrid between these two types bears the orbicular, non-lobed, majus type of leaf, but its leaves are notably larger than those of either parent. In the F_2 generation a third type appears. This type has the same general pattern as the lobed leaves of *T. peltophorum fimbriatum*, but instead of rounded lobes it has acute lobes and angular sinuses (Figure 9*B*). Segregation in the F_2 generation gave 196 round, 51 roundly lobed, and 14 acutely lobed leaves. This segregation closely conforms to a 12:3:1 ratio, with a p value of .88 indicating the presence of two genes in epistatic relationship. The results of backcrosses support such an assumption. The two genes may be designated by *L-l* and *U-u*. *L* produces orbicular leaves, and *U* in the absence of *L* produces roundly lobed leaves. Individuals which are *lu* have acutely lobed leaves. Thus *LU* and *Lu* individuals are orbicular, *lU* roundly lobed, and *lu* acutely lobed.

By developmental analysis it has been possible to obtain some idea as to how these factors controlling leaf shape operate. The youngest leaves in all the peltate-leaved species of Tropaeolum have about the same shape and are all miniatures of the mature leaves of plants with acutely lobed leaves (*lu*) (Figure 9*A*). There is some variation in the proportions, the ratio of length to width being generally greater in the younger leaves, but in every instance the pattern is identical. This together with the fact that the deeply lobed leaves are smaller than the unlobed suggests that the shape of the leaf may be in part dependent on the size to which it grows. The existence of such a relationship between size and shape is emphasized by measurements of dimensions in mature leaves of the various types: (Table I).

It is evident from Table I that there is an inverse relationship between the

*Massachusetts State College, Amherst, Massachusetts. Department of Botany, Columbia University, New York City.



NASTURTIUM LEAF TYPES

Figure 9

A—Juvenile leaf. The adult leaf form is conditioned by two pairs of genes, *L,l* and *U,u*. The types encountered are: *B*—Acutely lobed leaf (*lu*). *C*—Roundly lobed leaf (*Lu* or *lU*). *D*—Round leaf (*LU*). The figures represent average size leaves of each type. A × 3/5, B, C, D, × 2/3.

size of the leaf and the depth of the lobing. This immediately poses the question as to whether the small acutely lobed leaves (*lu*) may not be merely persistent juvenile forms. That this is not so is indicated by the fact that the round leaves have already assumed their round shape when they are still considerably smaller than the mature size of the acutely lobed leaves. This means that the genes *L* and *U* affect not only absolute growth but also relative growth.

If a young leaf which is acutely lobed is to become rounded in outline when mature it is apparent that the tissue in the sinuses must grow either more rapidly or for a longer period of time than at the apices. Which of these two alternatives obtains is not immediately indicated by the data presented here, but it is apparent that when the gene *U* is present there is relatively more growth than with *u*, either for a longer period of time or at a more rapid rate, in the sinuses than at the apices, hence the pointed apices are transformed into rounded lobes. When the gene *L* is present there is an even greater growth of the sinus tissue relative to that of the apices, enough more in fact to cause the whole leaf to become rounded. When both *L* and *U* are present the sinus tissue actually extends further than that of the original apices. It is of particular interest to note that the increase in size of the leaves of the F_1 hybrid between *Lu* and *lU* type plants, originally thought to be due to hybrid vigor, resolves itself simply into a combination of two genes.

The relative rates of growth of the various types of leaves must be left to a further study, but it is clear that the genes *U* and *L* produce respectively greater total growth, and that in each instance the greater proportion of the

TABLE I.—Measurements of Tropaeolum Leaves

Genotype	Leaf size, average of 100 leaves	
	Length (cm)	Width (cm)
LU	7.49±.09	8.32±.15
Lu	6.69±.09	7.29±.11
lU	5.59±.07	5.25±.06
lu	4.27±.12	4.63±.10

growth produced takes place in the incised portions of the leaf.

Color. — Rasmuson[7] has found dark green color in the leaves of Tropaeolum to be determined by two dominant genes, the recessive allele of either of which results in the expression of a lighter (medium green) type. He states further that both medium green and dark green are dominant to yellowish green, also that both are dominant to the variegated condition, a green-white mosaic type. Whaley and French[13] found Rasmuson's conclusions to be essentially correct. A cross between two medium-green-leaved types produces a hybrid darker than either parent. Upon selfing the hybrid a population of 147 dark green, 96 medium green, and 18 pale green plants was produced. The distribution represents an excellent fit to a 9:6:1 ratio indicating two complementary factors.

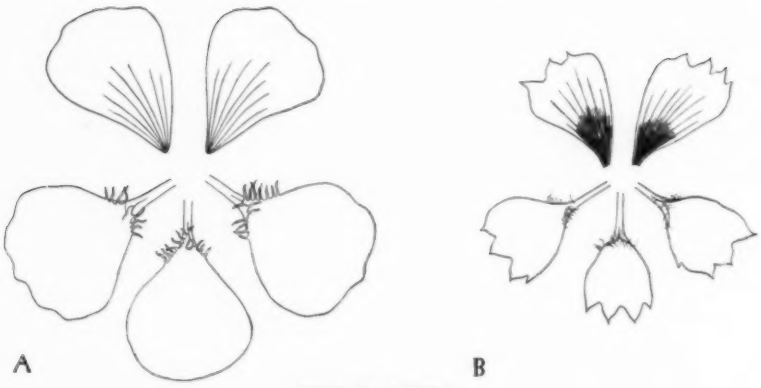
In determining leaf color several leaves of each individual plant were carefully matched against Ridgeway's color standard[10]. When this was done it became apparent that there are several genes in addition to the two basic ones concerned in the ultimate expression of leaf color, for while there is comparatively sharp segregation into three distinct classes there is wide variation within the classes as to tone and intensity. There are obviously many secondary factors such as the distribution of anthocyanin and other pigments, and the relative degree of pubescence which affect the final expression of color. It is significant, however, that there seems to be no correlation between the presence of anthocyanin in the rest of the plant and the expression of the darker greens in the leaves. The fundamental differences appear to be confined to the chlorophyll or its closely associated pigments.

The Flower

Petal number. — Garden varieties of nasturtiums bearing double flowers have been receiving considerable attention from commercial flower growers in recent years. Doubling of flowers in certain species of Tropaeolum has been known for more than two hundred years. The usual type of doubling consists of a proliferation of both the upper and lower petals accompanied by occasional petaloidy of the stamens. The number of petals produced in a double flower ranges from 6 to 15, the number varying within the flowers of an individual plant as well as among plants. There is a considerable tendency for the degree of doubleness to increase as plants become older. In many cases several single flowers will be produced by a plant before the doubleness becomes apparent. Eyster[2] and Eyster and Burpee[3] found that doubleness is recessive to the normal single flowered or five-petaled type, and that a single gene is concerned. The results in the present experiment support this conclusion. The hybrid between a single flowered and a double flowered type is single flowered, and the distribution in the F_2 generation is 196 single flowered and 65 double flowered individuals. This represents an excellent fit to a 3:1 ratio. The backcross data agree with this result.

Petal Shape. — In the genus Tropaeolum there are two common petal types; that characteristic of the common garden nasturtium, simple rounded and entire or divided by a small cleft into two rather indistinct lobes (Figure 10*A*), and a dentate or somewhat digitately dissected type in which the amount of dissection varies among species and varieties (Figure 10*B*). There are some forms in which both types of petal occur in the same flower, the three lower members being of one type, and the two upper members of the other type. In these, however, the degree of dentation of petals is small, the number of teeth remaining small and the incisions shallow. In addition, there are some forms in which there is a suppression of some of the petals as in *T. peregrinum*. Members of the species majus are characterized by having the rounded petal type; *T. peltophorum fimbriatum*, by the serrate type. The hybrid between the two has the rounded type indicating its dominance. In the F_2 population the parental types reappear in the ratio of 188 rounded to 73 serrate. This character is further discussed later in this paper.

Petal Color. — Flower color in the



PETAL TYPES

Figure 10

A—Entire petal type, characteristic of *T. majus*. *B*—Dentate petal type, characteristic of *T. peltophorum fimbriatum*. The figures represent average size petals of each type.

genus Tropaeolum has long been of interest because of the wide range of colors and their various patterns, and because of several complications concerned with its expression. Weiss[12] reported the occurrence of a plant which bore both yellow and red flowers. The offspring from selfing consisted of both yellow and red flowered individuals, and one which bore both yellow and red like the original parent. The variation seemed to be caused by the weather. Rawson[8,9] reports permanent genetic variations in color produced by light.

The extent of the color range is made possible by the occurrence of both plastid and cell sap colors in most of the types. The plastid colors range from very pale yellow to deep orange and are visible only when the anthocyanin pigments are absent. The anthocyanin (sap) colors range from pink through various reds to a dark maroon. There are also some modifications which tend toward magenta and bluish tints. The reds are further modified by the nature of the yellow coloration of the flowers. As a rule reds are darker in the absence of a well developed bright yellow pigment and more orange in its presence. The inheritance of the two types of coloration appears to be completely independent.

Rasmuson[7] states that there is a monogenic difference between light and dark yellow, both being due to plastid pigments; likewise between the presence of red (anthocyanin) and its absence (yellow). In the latter case he finds the homozygotes to be dark red, the heterozygotes light red, the recessives being of course vellow. Variegated flowers are dominant to self-colored types. Eyster and Robinson[4] found that the anthocyanin colors are more or less epistatic over the plastid colors.

The results in the present experiment tend to clarify the color inheritance situation somewhat. The female parent used had a pure yellow flower color, termed "cadmium yellow" by Ridgway.[10]. The pollen parent was a pure red. The F_1 hybrid was red, though somewhat lighter than the red parent. In the F_2 generation there appeared a whole range of colors; two shades of pure yellow; a red spotted, yellow type; a series of flushed red types, in which more or less red color is diffused over much of the petal surface; and two shades of pure red.

The situation becomes somewhat more clear when the types of color expression are considered separately. In the F_2 generation of the present experiment there appeared 65 yellow individuals. Of these 61 were dark yellow in color the same as the parent type. The re-

maining 4 were a pale cream yellow. The distribution suggests at once a case of duplicate factors with the double recessive resulting in the pale yellow condition. The fit to a 15:1 ratio is very close ($p = .96$). It is possible further to distinguish between the yellow types in those flowers where the yellow is partially obscured by red, and the distribution fits the same ratio. There is some indication in the data of Shaw[11] that there may be several more factors concerned with the intensification of the yellow plastid colors. There are at least three intermediate stages between the palest and the most intense yellow, and the results of his crosses indicate that there may be as many genes concerned in the intensification of the color. The basic color, then, is a yellow plastid pigment and the intensity of its expression appears to be controlled by at least two and possibly four or five genes.

As has been stated, a cross between a pure red and a yellow produces a red F_1. Upon selfing, red (indicating flowers with any anthocyanin at all present), and yellow (absence of red), segregate in the proportion of 196 red to 65 yellow, a simple monogenic ratio indicating that there is one basic gene concerned in the expression of red sap color. Since all the homozygous recessives are yellow they need not be considered further. Among the 196 red plants there is further segregation into three distinct types, a pure red, a spotted type in which the red color is confined to a definite spot near the base of each petal, and a flushed or mottled type in which red color is more or less diffused over the petals. This last type is further notable in being very susceptible to environmental influences. Its expression is markedly affected by either light or temperature, perhaps both, and by the age of the plant. In the same plant the amount of red apparent may be very minute or a whole petal with the exception of the claw fringe may be covered. In the course of a season it is not unusual for a single plant of this type to change its flower color from almost pure yellow to almost pure red. Even two adjacent flowers on a single plant may be one predominantly yellow and the other predominantly red.

It is possible to explain this rather involved situation quite simply if it is assumed that the action of four different genes is concerned. The basic gene for the production of red anthocyanin may be denoted as A_n. This gene A_n must be present in order to have any anthocyanin color produced. Three additional genes, M, M_1 and p may be supposed to be concerned in the expression of the red sap color. The complimentary genes M and M_1, when present together in individuals containing also A_n result in the production of a relatively large amount of red anthocyanin color. When in addition, the dominant gene P is present this anthocyanin color is distributed uniformly over the whole petal surface. When p, the recessive allele of P, is present in individuals containing A_n, M, and M_1, the red sap color is localized to a single spot at the base of each petal. The presence of either or both of the recessive alleles m or m_1 in individuals having also A_n results in the distribution of red color which is termed flushing. This condition is relatively unstable, the amount and the extent of red coloration varying with certain environmental factors. The action of the spotting gene p may or may not be discernible in individuals having flushed petals. According to this scheme the results obtained may be represented as follows:

TABLE II

P_1	A_nM_1MP	(red)		
P_2	a_nmm_1p	(yellow)		
F_1		$A_na_nMmM_1m_1Pp$		
			Theoretical	Actual
F_2	A_nMM_1P	(red)	$\frac{81}{256}$	81
	A_nMM_1p	(spotted)	$\frac{27}{256}$	26
	A_nMm_1P A_nMm_1p A_nmM_1P A_nmM_1p A_nmm_1P A_nmm_1p	(flushed)	$\frac{84}{256}$	89
	a_n, etc.	(yellow)	$\frac{64}{256}$	65
				261

The distribution in the F_2 population represents an excellent fit to the theoretical ratio ($p = .97$).

Fragrance.—An attempt was made to analyze the inheritance of fragrance. Using as one parent a type which possesses a high degree of fragrance and as the other one which possesses no fragrance at all, a hybrid was obtained which seemed intermediate between the two in degree of fragrance. Six persons were asked to classify each of the parents, the hybrid, and 109 second generation plants as (1) having intense fragrance, (2) having no fragrance at all, or (3) having some degree of fragrance. Blakeslee[1] has shown that there are differences in the ability of individuals to distinguish odors. This was found to be true among these six persons. The amount of time elapsing between tests also made for varying results. Further, either the human receptivity or the actual degree of fragrance appears to vary with the time of day and with atmospheric conditions as well as with the age of the flower. For these reasons no attempt is made at a genetic analysis, but the general conclusions of the group are presented as representing some indication of the manner of segregation. As has been stated, one parental type was fragrant to all six persons, the other not to any, and the hybrid more or less intermediate. The majority opinion rated 9 individuals in the F_2 generation as fragrant as the one parent, 72 as possessing some degree of fragrance but not so much as the fragrant parent, and 2 without any fragrance whatever. Reliable analysis of fragrance factors, however, requires a dependable means of detection and measurement.

Factor Interaction

From an analysis of a population which was admittedly too small, Whaley and French[13] suggested linkage between the factor controlling leaf shape and the one controlling petal shape. It was noted at the time that one of the recombination classes failed to occur, and supposing this to be due to the small size of the population a single crossover individual was assumed and the maximum possible crossover value was calculated. Further analysis indicates that this condition is probably subject to a different interpretation. If the inheritance of petal shape and leaf shape are considered independently they seem to conform to simple Mendelian schemes, one with a single factor concerned, the other with two factors in epistatic relationship. If, however, the two characters are considered in combination a curious condition becomes apparent. When a round-petaled, orbicular-leaved plant with the constitution *ELu* (*E* being the gene for round petals) was crossed with a dentate-petaled, lobed-leaved plant, *elU*, the hybrid was round-petaled with orbicular leaves and the following F_2 distribution was obtained:

TABLE III

Entire petals, orbicular leaves (*ELU*)	185
Dentate petals, orbicular leaves (*eLU*)	11
Entire petals, roundly lobed leaves (*ELu*, *ElU*)	3
Dentate petals, roundly lobed leaves (*eLu*, *elU*)	48
Entire petals, acutely lobed leaves (*Elu*)	0
Dentate petals, acutely lobed leaves (*elu*)	14

It is apparent that neither independent assortment nor any degree of linkage will explain such a distribution. The explanation seems rather to rest upon the developmental relationships of the genes in question. If it is assumed that a common factor is responsible for the dentation of petals and lobing of leaves, and the sequence of development from the acutely lobed to the orbicular form in the ontogeny of both petals and leaves is considered, it is possible to explain the situation. It has been supposed that the genes *L* and *U* increase growth of the leaves, and at the same time bring about an earlier obliteration of the juvenile lobed form. It seems very probable that the same situation may prevail in the case of the petals, varied somewhat by the fact that the ontogeny of the petal is later than that of the leaf. Such a supposition regards the association of entire petals with orbicular leaves, and dentate petals with lobed leaves to be the normal condition and explains the occasional appearance of dentate petals among the

orbicular-leaved individuals as the result of a partial delay or disruption in the development which normally obliterates the dentate form. The presence of a few entire petals among the roundly lobed leaf types may also be due to some minor variation in developmental rates. Unfortunately no note was made as to whether these few individuals had leaves which were any less lobed in form than the usual type. No entire petals occur in plants with acutely lobed leaves since such plants lack the genes *L* and *U* which are necessary if sufficient growth to obliterate the dentate form is to take place. That this is probably the case is evidenced by the fact that 100 petals of the dentate type average only 1.8 cm. in width against 2.9 cm. for the entire type, and length varies accordingly. The petal types then bear the same general size relationship to one another as do the leaf types. It is possible that further study of the condition may furnish evidence both for the method of development and for the concept of homology of petals and leaves.

Summary

The expression of acutely lobed, roundly lobed, and orbicular leaf shapes in Tropaeolum is controlled by two genes, *L* and *U*, in epistatic relationship. The factors appear to determine shape by controlling the amount and relationship of growth which takes place. The ultimate expression of leaf shape and petal shape is found to be due to certain definite developmental relationships.

Leaf color is dependent upon the presence of two complementary genes *G* and G_1; the presence of both resulting in dark green leaves; the absence of either, medium green; and the absence of both, light green color.

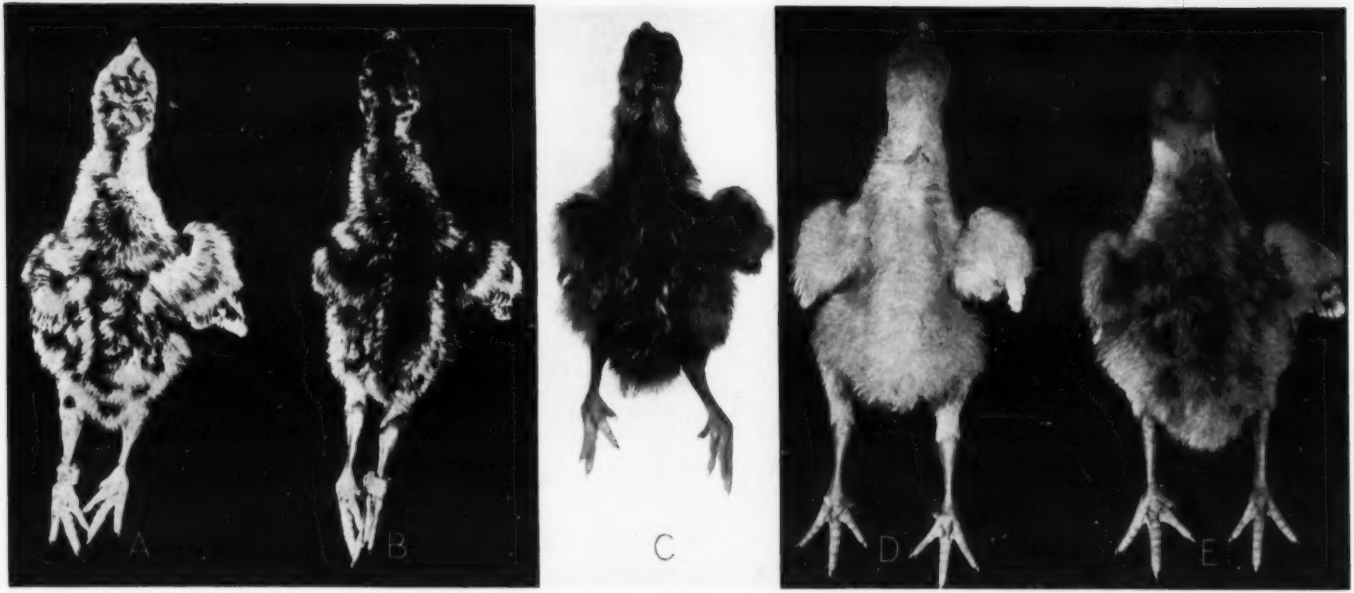
Yellow plastids form the basic color in most species of Tropaeolum. The intensity of the yellow color is dependent upon the cumulative action of at least two and possibly more genes. Red anthocyanin may accompany the yellow pigments. Its presence depends upon one gene A_n, its intensity upon two more, *M* and M_1, and localization into a definite pattern upon a fourth factor, *p*.

Petal number is inherited on a simple monogenic basis with the normal five-petaled type being dominant to the simple double type.

The writer wishes to express his appreciation to the Massachusetts State College for financial assistance which made this study possible. He also wishes to acknowledge his indebtedness to Professors A. P. French and R. A. Van Meter of the Massachusetts State College and to Professor E. W. Sinnott of Columbia University for valuable advice and criticasm.

Literature Cited

1. Blakeslee, A. F. Unlike reactions of different individuals to fragrance in Verbena flowers. *Science* 48:298-299. 1918.
2. Eyster, W. H. Inheritance of doubleness in Tropaeolum majus. (Abstract.) *Amer. Nat.* 70:49. 1936.
3. ———— and D. Burpee. Inheritance of doubleness in the flowers of the Nasturtium. *Jour. Hered.* 27:51-60. 1936.
4. ———— and B. A. Robinson. Genetic studies of Tropaeolum. (Abstract.) *Amer. Nat.* 70:49. 1936.
5. Fischer, H. Ein interessanter Tropaeolum Bastard. *Gartenflora* 62:278-282. 1913.
6. McLean, F. T. A genetic analysis of the inheritance of fragrance of gladiolus. *Bull. Torrey Bot. Club* 65:181-198. 1938.
7. Rasmuson, H. Die Hauptergebnisse von einigen genetischen Versuchen mit verschiedenen Formen von Tropaeolum, Clarkia and Impatiens. *Hereditas* 1:270-276. 1920.
8. Rawson, H. E. Color changes in flowers produced by controlling insolations. *Rep. Brit. Assn.* 902-903. 1908.
9. ————. Variation of structure and color of flowers under insolation. *Rep. Brit. Assn.* 711-713. 1913.
10. Ridgway, R. Color Standards and Color Nomenclature. Washington, D. C. 1912.
11. Shaw, J. K. Massachusetts State College, Amherst, Massachusetts. (Unpublished.)
12. Weiss, F. E. The genetics of a Tropaeolum mutant. *Rep. Brit. Assn. Adv. Sci.* 1928:616-617. 1929.
13. Whaley, W. G., and A. P. French. A genetic study of Tropaeolum. *Proc. Am. Soc. Hort. Sci.* 34:598-602. 1937.



DOWN PATTERNS OF TURKEY COLOR VARIETIES

Figure 11

A, Black-winged bronze; *B*, Bronze; *C*, Narragansett; *D*, White with some buff, particularly on the head; *E*, Bourbon red. These types were used in the analysis of the wing barring factor in the turkey.

INHERITED NON-BARRING OF THE FLIGHT FEATHERS IN TURKEYS

V. S. Asmundson
University of California, Davis

IN the autumn of 1935 one of my students, Mr. George Nicholas of Petaluma, California, called my attention to birds in his flock with plumage color differing from that of their Bronze turkey parents. These birds, both males and females, had black primary flight feathers and black secondary flight feathers with white tips. There were also white markings in the region of the shoulder. These were present on more feathers in the female than in the male. The breast feathers of the females had an irregular white line near the tip instead of the white tip seen on the breast feathers of Bronze adult females. The saddle and tail feathers were marked exactly as on the Bronze (Figure 12). To simplify the discussion in this paper, birds with this plumage pattern will be called black-winged bronze, as distinguished from the typical Bronze with barred flight feathers.

A few of these black-winged bronze were mated together the following spring. All the poults from this mating had the same down pattern, which resembled the Bronze in the distribution of the dark pigment. There was, however, much less black and brown pigment, whereas more of the down was nearly white, so that the poults were considerably lighter in color than the Bronze. The juvenile plumage was also lighter colored than that of the Bronze. The down patterns of the black-winged bronze and of the other varieties used are shown in Figure 11.

Experimental Matings

The black-winged bronze were mated with the Bourbon Red, Narragansett, and white turkeys. The white turkeys used were all from a single white hen that segregated out of a flock of Bronze. The black-winged bronze was not mated to the Bronze, since the matings made were expected to show how the new color variety differs genetically from the Bronze.

Black-winged Bronze × Bourbon Red. The F_1 generation from the black-winged bronze mated to the Bourbon Reds all had approximately the same pattern, regardless of which way the cross was made. In down and plumage the hybrids resembled birds obtained when the Bronze are mated to the Bourbon Reds, except that the down of the latter is slightly darker. As has been shown elsewhere,[1] the F_1 poults resemble the Bronze closely in the distribution of pigment in the down. Being much browner, however, they are readily distinguished from the Bronze and from the Bourbon Reds. The brownish color of the down characteristic of these F_1 poults has been designated the F_1 pattern.

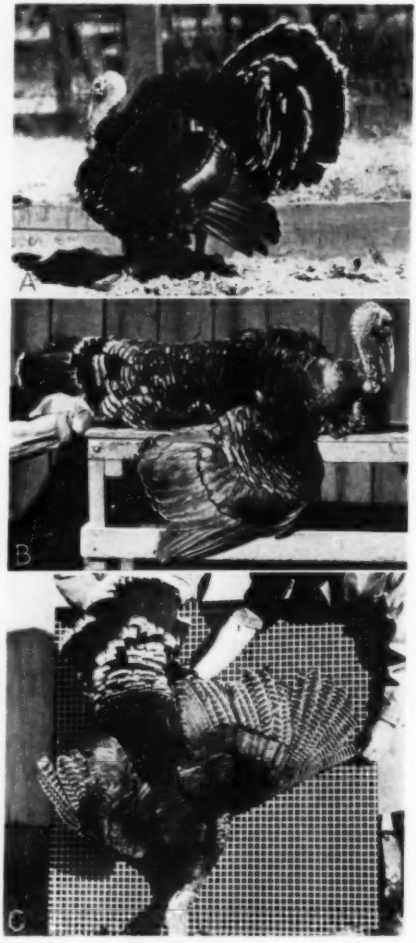
The down patterns of the F_2 generation from black-winged bronze mated to Bourbon Reds are shown in Table I. The data can be interpreted on the basis of two pairs of autosomal genes. In the terminology suggested by Robertson[4] for the Bronze and Bourbon Red patterns, the black-winged bronze have the constitution *RR*. The nonbarred wing is determined by a recessive autosomal gene *l*. The Bourbon Red carries the dominant gene *L*, but barring is not expressed with the Bourbon Red pattern. The genetic constitution of these two plumage color patterns, as shown by the results of reciprocal matings between them, is then as follows: black-winged bronze, *RRll*; Bourbon Red, *rrLL*.

The results shown in Table 1 show the number of poults with each of the different down patterns obtained except for seven birds with light Bourbon Red down which were grouped with poults of

darker shade having the same pattern. Six poults with pure white down were assumed to be double recessives, homozygous for non-barred flight feathers *ll* and for Bourbon Red plumage *rr*. Breeding tests of such birds indicate that this is correct since 26 progeny with pure white and 2 with doubtful down color were obtained. The eight that hatched all had pure white down and later developed red plumage. Such birds have lighter colored plumage than typical Bourbon Reds.

The numbers of poults obtained (Table 1) deviate from the expected in three classes, namely, an excess of bronze, a deficiency of a new plumage-color combination—F_1 with non-barred wings (Figure 13*A-B*) —and a deficiency of pure white poults, with the result that P = .01. Breeding tests of the birds having the F_1 with non-barred wings pattern gave 29 black-winged bronze, 52 F_1 with non-barred wings and 27 pure white down, in good agreement with the number expected on the basis of 1 *RRll* (black-winged bronze) : 2 *Rrll* (F_1 with non-barred wings) : 1 *rrll* (pure white down, red plumage). The number (Table 1) of barred-winged birds is 131 : 34 non-barred-winged birds (omitting birds with red or white down and red plumage), where the expected number is 123.75 : 41.25. This is a good fit to the expected ratio of three birds with barring to one bird without barring. The proportions of Bronze to F_1 pattern to Red obtained is 72 : 93 : 46, a poor fit (P = .01) where the number expected on the basis of a 1 : 2 : 1 ratio is 52.8 : 105.6 : 52.8. In spite of these deviations from the expected results which are traceable to the three classes mentioned above, the interpretation given in Table 1 undoubtedly gives the correct genotypes for the various phenotypes shown.

Black-winged Bronze × *White*. The F_1 generation from the cross, black-winged bronze ♂ × White ♀ ♀ and the reciprocal all had the Bronze pattern. The hybrid adults could usually be distinguished from pure Bronze by white edging and by a wide, pure white tip on the secondaries. The down color of the hybrid poults was slightly more greyish and the striping less distinct than that of Bronze poults. The white birds used



ADULT PATTERNS

Figure 12

A—Black-winged bronze male with non-barrel flight feathers and with a little grey on the shoulders. Otherwise the plumage color is the same as that of the Bronze variety. *B*—Black-winged bronze male with non-barred flight feathers. Note white tips on secondary flight feathers and grey feathers on the shoulders. *C*—Bronze male. Note barred flight feathers. Compare with *A* and *B*.

BARRED AND NON-BARRED FLIGHT FEATHERS

Figure 13

A-B.—Birds having the constitution *Rr* (F_1 pattern) out of Bourbon Red mated with Bronze, black-winged bronze, or Narragansett; *A*, with barred flights; *B*, with non-barred flights but with some stippling on the secondaries. It should be kept in mind that the birds such as those shown at *B* do not occur in the F_1 generation, but segregate out in the F_2 generation (see text and Table 1). *C*—Narragansett; *D*, Non-barred flight feathers on an otherwise typical Narragansett.

were, as stated above, descended from a white hen that had segregated out of a flock of Bronze. Obviously, the results might have been different had another strain of white turkeys been used, although when these white turkeys were mated to White Holland, all the progeny were white. Robertson[4] used the symbol *w* for the gene for white plumage, which he found to be a simple recessive to Bronze *W*.

The data for the F_2 generation are shown in Table II. The white turkeys evidently carried the gene *L* for barred wings and had the genetic constitution *wwLL*, whereas the black-winged bronze had the constitution *WWll*. Most White Holland and other white poults have brown down on the head and on all or part of the body, but show no striping. Poults with pure white down occur occasionally. There were at least six pure white poults in the F_2 generation, which were grouped with the other white poults in Table II. These pure white poults may have been homozygous for non-barred wing and therefore double recessives, but no breeding tests were made. It will be observed that there were actually far fewer white poults than would be expected. Since the value of *P* equals .02, only twice in 100 trials would so great a deviation be expected to occur through chance.

The small number of white poults may indicate the presence of a lethal gene or combination of genes. Other matings of the white turkeys do not, however, indicate that the white turkeys carry a lethal gene or genes. Thus when the white turkeys were mated to the Bronze, the F_1 generation were all bronze; the F_2 generation comprised 65 bronze to 16 white, a reasonably good fit for a 3 : 1 ratio. When the F_1's were backcrossed to the original white hen, three bronze and five white poults were obtained. Males and females occurred in approximately equal numbers among the bronze and white poults. These results agree with the assumption that the whites differ from the bronze by a single pair of autosomal genes, as suggested by Robertson,[4] white (*w*) being recessive to Bronze (*W*). All bronze varieties presumably have the constitution *WW*, since the results in the F_2 generation, with respect to white and colored, appear to be the same regardless of the colored variety used. Thus, in the F_1 generation, crosses of Bourbon Red with Whites gave all colored (F_1 pattern); in the F_2 generation, 149 colored to 46 white (expected on the basis of a 3:1 ratio, 146 colored to 49 white). Here again there is no indication of a lethal gene. Further matings are planned to test the various possibilities suggested by the data in Table II.

If it is assumed that Bronze and black-winged bronze occur in the numbers expected, the deficiency of white poults is actually greater than shown in Table II. Disregarding the white poults, the number of Bronze and black-winged bronze obtained agrees well with the 141 Bronze and 47 black-winged bronze expected on the basis of a 3 : 1 ratio.

Black-winged Bronze × *Narragansett*. A black-winged Bronze male was crossed with a Narragansett female. All the F_1's had Bronze down and plumage, but modified so that they resembled those obtained from the cross of black-winged bronze with white described above. Only a few F_2 birds were obtained. Those classified as to sex and plumage color were as follows: 2 Bronze (barred wings) females (Figure 13*C*); 1 black-winged bronze female; 1 non-barred wing Narragansett female (Figure 13*D*).

The reciprocal cross (Narragansett male × black-winged bronze females) gave 14 Bronze males and 15 Narragansett females. The down and plumage of the bronze F_1 was modified as already stated. The down of the F_1's with the Narragansett pattern was a little paler than that of pure Narragansetts; and the secondaries in the adult plumage were modified, showing white tips. The results for the F_2 generation (Table III) agree reasonably well ($P = .059$) with the results expected on the assumption that the Narragansett carries the dominant autosomal gene *L* for barring, whereas the black-winged bronze carries the recessive gene *l* for non-barred

flight feathers and the dominant sex-linked gene *N*. The Narragansett is known to carry the recessive sex-linked gene *n*.

Discussion of Results

A plumage pattern not previously described for turkeys is reported. Birds with this pattern (absence of barring on the flight feathers) have since been observed in other commercial flocks of Bronze and in a flock of supposedly pure wild Virginia turkeys (*Meleagris gallopavo silvestris*). The mutation, therefore, probably occurred a long time ago. Nevertheless, it has apparently not been reported in wild turkeys, although many subspecies differing but slightly from each other are now recognized by Bent,[2] Moore,[3] and others.

The results of the crosses made show that the Narragansett, as would be expected, carries the gene for barred flight feathers. A Narragansett with non-barred flight feathers has been obtained. Birds with such a pattern have not been observed in commercial flocks.

The gene *l* for non-barred flight feathers inhibits brown melanin in the down as shown particularly by the pure white down of birds homozygous for *l* and for *r*, the gene for red plumage color such as that of the Bourbon Red variety. The gene *l* may also produce a lighter-colored adult red plumage. The Bourbon Red carries the gene *L* for barred flight feathers but does not exhibit barring. Apparently the Bourbon Red differs from the Bronze and from some other colored varieties essentially in having brown instead of black melanin.

Judging from the results obtained, the gene for non-barred flight feathers is independent of the genes for colored plumage *W* and red plumage *r*. Presumably the only new plumage patterns that the *l* gene can be expected to produce are the ones with the Bronze (black-winged bronze) and Narragansett (Narragansett with non-barred flights). The new varieties are, of course, homozygous for the autosomal recessive gene *l*, whereas the Bronze and Narragansett have the constitution *LL* or *Ll*. As already stated, the heterozygotes, both Bronze and Narragansett, can usually be distinguished from the homozygotes by the more pronounced white tipping and edging on the secondary flight feathers. It may also, however, modify other plumage patterns such as the Bourbon Red by producing a different shade of color without changing the distribution of the pigment.*

Summary

Birds resembling the Bronze variety but having black primary flight feathers, black secondary flight feathers with white tips, and grey feathers on the shoulders were found in a commercial flock of Bronze turkeys. When mated together, these black-winged bronze birds bred true, the down pattern of the poults being much lighter than the down pattern of the Bronze.

Light down and non-barring of the flight feathers is determined by an autosomal gene *l* recessive to the gene for

Table I. F_2 generation from black-winged bronze mated to Bourbon Reds

Down pattern*	Number	Number recessive	Probable genotype
Bronze	50	50.6	WWLL, WWLl
F_1 pattern	79	79.2	WwLL, WwLl
Black-winged bronze	25	25.3	WwLl
F_2 with non-barred wings	19	18.4	WwLl
Bourbon Reds	60	59.6	rrLL, rrLl
Pure white	8	19.9	rrll
Totals	241	241.0	

*The down pattern corresponded to the bronze pattern except in the case of the poults with pure white down which had red plumage.

Table II. F_2 generation from all matings of black-winged bronze to white birds.

Down pattern	White ♂ x black-winged bronze ♀	Black-winged bronze ♂ x white ♀	Total numbers observed	Total numbers expected	Probable genotypes
	Number	Number	Number		
Bronze	93	40	179	134.3	WwLL, WwLl, WwLl, WwLl
Black-winged bronze	54	33	49	61.4	WwLl, WwLl
White	21	12	33	30.3	wwLL, wwLl, wwLl
Total	168	91	261		

Table III. F_2 generation from black-winged bronze female mated to Narragansett male.

Plumage pattern*	Number observed		Total sex unknown	Total	Numbers expected
	Males	Females			
Bronze	15	12	2	29	30
Narragansett	14	9	2	25	30
Black-winged bronze	10	9		19	20
Narragansett with unbarred flights	6	4		10	10
Totals	45	34	4	83	80

*The black-winged bronze and the Narragansett with unbarred flights had down patterns that were identical or so nearly alike that they could not be distinguished from each other.

*At the Seventh World's Poultry Congress in Cleveland, July 28 to August 7, 1939, a pair of birds having this black winged bronze pattern were exhibited under the name of Crimson Dawn.

darker down and barred flight feathers *L* carried by the Bronze, Narragansett, and Bourbon Red varieties and by some white birds. These genes appear to have little or no influence on the distribution of the brown melanin of the Bourbon Red but suppress brown melanin in the down of poults homozygous for *r* as proved by breeding tests with pure white poults (which later developed red plumage) and other birds obtained in the F_2 generation from the cross of black-winged bronze on Bourbon Reds.

Birds with the Narragansett pattern, except for non-barred flight feathers, were obtained in the F_2 generation from the cross with Narragansetts. These differ from the black-winged bronze in having the sex-linked recessive gene *n*, but both have light down *l* and are colored varieties *W* with black melanin *R*.

Literature Cited

1. Asmundson, V. S. Note on a Bronze-Bourbon Red mosaic. *Jour. Gen.* 45:25-30. 1937.

2. Bent, A. C. Life histories of North American gallinaceous birds; orders galliformes and columbiformes. *Bull. 162, U. S. Nat. Mus.* Pp. 1-490. 1932.

3. Moore, R. T. A new race of wild turkey. *Auk* 55:112-115. 1938.

4. Robertson, W. R. B. Revised terminology for the chief color factors concerned in crosses among the breeds of the turkey. *Anat. Rec.* 44:289. 1929.

✦

More Advice for the Growing-ups

"LIFE and Growth" is one of a series of books dealing with the intelligent adjustment of youth to its social environment.* It is offered by the Commission on Human Relations to aid young people of high school and junior college age. The book consists of three main parts—I, Human Life and Social Progress; II, The Individual and the Way He Grows; and III, New Life and Social Change. It is clearly, interestingly, and accurately written, emphasizing that "modern medicine, psychology, physiology, and biology are coming closer and closer together in agreement that man is a complex, intricately organized, interdependent organism. Coordination and regulation of internal and external factors are perhaps the most intricate and the most significant of human functions."

The second part of the book deals with the problems of sex development and sex function in an able, frank, and straightforward manner. Although it lays more than the usual stress on the effect of the internal secretions on growth and development, it fails to mention the formation of the corpus luteum. The book is concluded with a forward-looking chapter in which belief in the worth of human personality and its capacity to grow and develop is suggested as the new faith of American youth so essential to a successful democracy.

Suggestions for further reading seem well selected. However, some of the books on sex relations presuppose a considerable amount of sophistication on the part of high school students.—G. HAINES.

*Keliher, A. V. *Life and Growth*, pp. 245, 36 illus. New York: Appleton-Century Co., 1938.

DIFFERENCES IN INCIDENCE OF ENCEPHALOMYELITIS IN HORSES*

W. V. Lambert, S. R. Speelman, and E. B. Osborn†

INHERENT differences in resistance to certain types of diseases are known to exist in both animals and plants. Among animals, differences in resistance to bacterial and virus diseases have been shown by Hagedoorn-LaBrand and Hagedoorn[2], Irwin[3], Schott[6], Lambert[4], Webster[7], Wright and Lewis[8], Gowen and Schott[1], and others. In plants such differences have been extensively studied and the development of resistant strains has become an effective means for combatting certain plant diseases. Such differences are relative rather than absolute, the degree of resistance depending upon such factors as the inborn qualities of the host, the virulence of the pathogenic agent, the environment, age, and physiological condition of the host, degree of infection, et cetera.

In the larger animals, species differences in resistance to certain diseases are well known but few well-authenticated cases of such intra-species differences are known. In horses the opportunities for making such observations are rare and it is believed that the data reported herein, describing a differential resistance to encephalomyelitis, will be of definite interest to biologists, horsemen, and veterinarians alike.

Materials Used

These records were obtained from the experimental stud maintained at the United States Range Livestock Experiment Station at Miles Ciy, Montana. The 221 horses in this stud represented several types of breeding but the largest percentage consisted of grade Belgians and grade Thoroughbreds. In addition, there were a few Morgans, Nonius, and some crosses from Nonius stallions on grade Belgian and Thoroughbred mares. Horses of various ages were represented in most groups, the frequencies for each group and age being shown in Table I.

In July of 1938 a rather severe epizoötic of encephalomyelitis occurred in this stud, 30 of the 221 animals being affected. It is on the basis of these cases that the conclusions reported herein are based.

It is generally accepted that encephalomyelitis is transmitted by biting insects such as mosquitoes[5] and not from direct contact with infected horses. No data are available on the source of or method of spread of the disease in this epizoötic. Since the horses of the different breeding groups were distributed at random in the various pastures of the station, the opportunities for exposure to the disease were equal between the groups in which the incidence of the disease is compared.

Incidence of Disease by Breed Groups

The incidence of encephalomyelitis for the various types of breeding and for the different age groups in each type are shown in Table I. Of the 221 horses in the stud 31 were either purebred Nonius or the result of crosses of Nonius stallions on the grade Belgian or Thoroughbred mares. Of this group, 18 developed encephalomyelitis, this being 58.1 per cent of the animals of this breeding. Among the 190 animals of

*U. S. Department of Agriculture in cooperation with Montana Agricultural Experiment Station.

†Respectively, Senior Animal Husbandman, in Charge of Genetics; Associate Animal Husbandman, Acting in Charge of Horse and Mule Investigations; and Junior Animal Husbandman, U. S. Range Livestock Experiment Station, Bureau of Animal Industry.

all other groups only 12, or 6.3 per cent of the total, were affected. However, a comparison on the basis of totals is somewhat misleading for, as is apparent from Table I, the higher incidence of the disease occurred among the younger animals (yearlings and foals). Since all animals with Nonius breeding were yearlings or foals, except five mature animals imported from Hungary in 1935, a comparison of the incidence of the disease in these two age groups has been made. Among 41 foals 22 were of Nonius breeding and 19 of other breeding. Of the 22 foals of Nonius breeding 16, or 72.7 per cent, were affected, whereas only 2 out of 19, or 10.5, of the foals of other breeding were affected. This difference is highly significant, P being less than .01.

Only four yearlings with Nonius breeding were in the stud but two of these were affected, while among 27 yearlings of other breeding only three were affected. Here also the trend in mortality was in the same direction as was observed in the foals. Thus it is apparent that in these two comparable age groups the horses with Nonius breeding were more susceptible to this strain of encephalomyelitis virus.

Of the 30 animals affected seven died. Three died during the course of the disease and four died during the convalescent period. Of the latter cases one apparently recovered but was found dead in the pasture the latter part of September. Of the others, according to the veterinarian in charge, two died from the effects of the disease and one from septicemia resulting from bed sores acquired during paralysis. Six of the seven animals that died were of Nonius breeding, five of these being foals and one a yearling. The seventh animal, a yearling, was the one found in the pasture in late September.

The distribution of the affected animals in the various pastures both preceding and during the outbreak of the disease precludes the possibility of the differences in resistance observed in the various groups being due to their location on the station. The station comprises an area of approximately 50,000 acres and the various pastures are well scattered over most of this area. These pastures varied in size from about 400 acres to several thousand acres. The distribution of animals of the various groups in these pastures was largely at random so that all groups had similar chances of exposure to the disease.

During the breeding season, from June 1 to July 15, 70 head of mares (including the purebred Nonius) with 39 foals were rotated in pastures near the station headquarters. The density of the stocking in these pastures was greater than in the other pastures but since all mares with foals were included the opportunities for exposure in the various groups were equal. Twenty-four of the 30 cases of encephalomyelitis occurred in this group.

The distribution of the cases of encephalomyelitis in the various pastures gives additional support to the above conclusion. Encephalomyelitis occurred in each of the several pastures and in most of the pastures animals from two or more of the different groups were affected.

Fourteen mares of the grade Belgian stock, all suckling foals, were vaccinated with brain emulsion vaccine on June 18 and June 26. Of these mares, one was affected with encephalomyelitis on July 13, this being the first case of encephalomyelitis in the 1938 epizoötic. The foal of this mare likewise developed encephalomyelitis, as did the foals of three others of these mares. The incidence of infection in the vaccinated mares (one out of 14) was equally as high as that observed in the unvaccinated mature stock. Three of the four foals were from a cross with a Nonius stallion and one from a purebred Belgian stallion.

Incidence in Various Age Groups

The distribution of animals of the various ages in the stud is shown in Table I. It is apparent that the younger animals were somewhat more susceptible to the disease than the older animals. A fair comparison of the in-

fluence of age on susceptibility can be made only for the groups not involving Nonius or crossbred-Nonius breeding since, aside from the five purebred mature Nonius horses, only yearlings and foals of this breeding were represented.

It would appear that the younger animals of other than Nonius breeding were somewhat more susceptible than the older ones, although the incidence of infection was so low in all of these groups that none of the differences are statistically significant. This is in line, however, with previously reported observations on age differences in resistance. No sex difference in the incidence of the disease was noted, as 16 of the animals affected were females and 14 males.

Previous History at the Experiment Station

Only one other outbreak of encephalomyelitis has occurred at the Range Station. The disease first appeared in the summer of 1937, and five animals were affected between the dates of August 12 and September 5. One of these animals was a mature Nonius stallion, two were grade Belgians, one a grade Thoroughbred, and one was a purebred Belgian. Three of these animals died, one of them being the Nonius stallion, Longfellow. At that time only ten animals with Nonius ancestry were on hand. Nine of these were purebred Nonius and one by a Nonius stallion and out of a grade Belgian mare. Previous to the time of his death, Longfellow had bred a number of mares. He was the sire of ten horses with Nonius ancestry that were in the stud at the time of outbreak of the 1938 epizoötic, and eight of these were affected. Sixteen of the other horses of Nonius breeding were sired by the stallion Shorty, and ten of these were affected. The other Nonius animals, all imported, were sired by several different stallions.

Discussion

From the foregoing facts it would appear that susceptibility to encephalomyelitis virus of the western type responsible for this outbreak is dominant to resistance, for the crossbred progeny from the Nonius stallions appeared about equally as susceptible as did the purebred Nonius of the same age groups. The reasons for the greater susceptibility of horses of Nonius ancestry are not definitely known. Perhaps there has been some natural selection for resistance in the horses that have been native to America for some generations for there is evidence pointing to the existence of virus-induced encephalomyelitis in this country for decades. The Nonius stock previous to its importation was not subjected to such selection for American-type encephalomyelitis virus is non-existent in Europe.

If resistance were recessive, natural selection would be favorable to the development of resistant strains, but the failure of horses in different vicinities to be exposed to the disease might explain the failure for the complete elimination of susceptibility from a stock. Whether there would be a differential selection between the eastern and western type viruses is unknown. These data do not throw any light on this point because most of the horses in this stud were descendants of horses bred at this station or at other localities in the western part of the United States. Because of the limited data and the absence of critical tests designed to answer some of these specific questions, broad generalizations on the rôle of previous selection on resistance and susceptibiltiy, and on the dominance of susceptibility are unjustified. They do, however, point to the fact that a differential resistance to encephalomyelitis exists in horses.

The fact that such differences exist should be of considerable interest and importance to the epizoötiologist, for such differences might account for a considerable part of the variation in mortality that is encountered in different outbreaks of the disease. Differential resistance of the type encountered here also might influence the response

to treatment with vaccines, though no evidence to this effect now exists.

It is hoped that these observations may stimulate others of a similar nature, for it is largely from such observations that a knowledge of the rôle of inheritance in relation to resistance or susceptibility to disease can be established in species such as the horse.

Summary

Statistically significant group differences in resistance to encephalomyelitis that were observed in an epizoötic occurring in 1938 in the stud of horses at the U. S. Range Livestock Experiment Station are reported. A greater percentage of horses of Nonius breeding were affected than those of other breeding. Among all yearlings 50.0 per cent of Nonius breeding were affected, whereas only 11.1 per cent of the yearlings of other breeding were affected. In the foals the corresponding percentages were 72.7 and 10.5. The differences observed were not attributable to the location of the horses on the station, their distribution being such that all groups had equal opportunity to be exposed to the disease.

The data confirm previously reported observations on age differences in resistance, the younger animals being the more susceptible. No differences in resistance were observed between males and females. Possible reasons for the differences observed are discussed.

Literature Cited

1. Gowen, J. W., and R. G. Schott. Genetic constitution in mice as differentiated by two diseases, pseudorabies and mouse typhoid. *Amer. Jour. Hyg.* 18:674-687. 1933.

2. Hagedoorn-LaBrand, A. C., and A. L. Hagedoorn. Inherited predisposition for a bacterial disease. *Amer. Nat.* 54:368-375. 1920.

3. Irwin, M. R. The inheritance of resistance to the Danysz bacillus in the rat. *Genetics* 14:337-365. 1929.

4. Lambert, W. V. Natural resistance to disease in the chicken. *Jour. Immun.* 23:229-260. 1932.

5. Schoening, H. W. Recent developments in equine encephalomyelitis. Address before 19th Annual Meeting Horse and Mule Association of America, Chicago, Ill. Leaflet 234, Horse and Mule Assoc. America: 3-13. 1938.

6. Schott, R. G. The inheritance of resistance to Salmonella aertrycke in various strains of mice. *Genetics* 17:203-229. 1932.

7. Webster, L. T. Inheritance of resistance of mice to enteric bacterial and neurotropic infections. *Jour. Exp. Med.* 65:261-286. 1937.

8. Wright, Sewall, and P. A. Lewis. Factors in the resistance of the guinea pig to tuberculosis, with especial regard to inbreeding and heredity. *Amer. Nat.* 55:20-50. 1921.

Table 1. The incidence of encephalomyelitis by breeding and age groups resulting from an outbreak among horses at the United States Range Livestock Experiment Station in 1938.

Breeding	Age groups								Total	
	3 years and over		2-3 years		1-2 years		Foals			
	Total no.	No. affected	Total no.	No. affected	Total no.	No. affected	Total no.	No. affected	No. animals	No. affected
Nonius	5	0	0	0	3	1	4	3	12	4
Nonius x Belgian	0	0	0	0	1	1	7	5	8	6
Nonius x Thoroughbred	0	0	0	0	0	0	11	8	11	8
Morgan	8	1	0	0	0	0	0	0	8	1
Belgian	60	1	9	1	9	2	9	1	87	5
Thoroughbred	54	4	13	0	18	1	10	1	95	6
Total	127	6	22	1	31	5	41	18	221	30

MANNOSE—
A New Taste "Thrill"

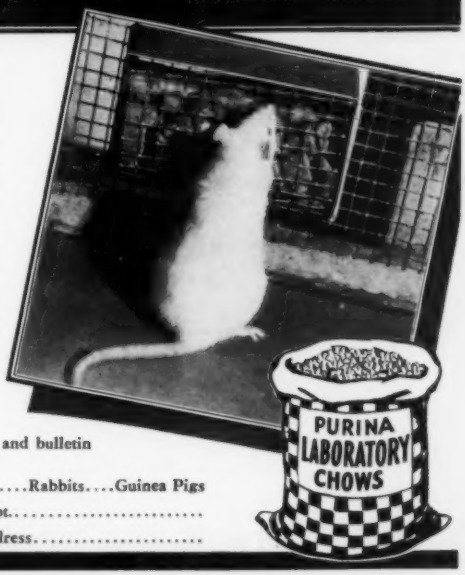
Six (more or less) delicious flavors in one pill — depending on who eats it. Adds interest and variety to the P. T. C. "Taster Test" which has been standard for several years to demonstrate inherited differences in taste perception. Send ten cents for a sample.

AMERICAN GENETIC ASSOCIATION

Victor Building **Washington, D. C.**

TRIED and TESTED

To Help Improve Results



IN THE CONSTANT search for ways to improve the results obtained with Purina Laboratory Chows, controlled feeding tests on rats, mice, rabbits and guinea pigs are run in the Purina Biological Laboratory. These tests act as a check on the results that Purina Laboratory Chows will give. This means that when you feed Purina Laboratory Chows you are feeding rations that have been developed as a result of this experimental work—rations that have been proved in actual controlled feeding tests on laboratory animals!

——————TEAR HERE——————

PURINA MILLS,
922 Checkerboard Square, St. Louis, Mo.

Please send sample of diet for following animals and bulletin giving ingredients, cost and feeding directions.

I keep about (No.).....Rats.....Mice.....Dogs.....Rabbits....Guinea Pigs

Name.....Dept.....

Institution.....Address.....

"HEREDITY" LANTERN SLIDES

MAN

16-397. Gregor Mendel's Handwriting — First Page of "Studies of Plant Hybrids".
17-435. Merton House—Home of William Bateson.
21-1-Cover. The "Fuchsia-Picture" of Mendel.
21-No. 2-Cover. Francis Galton.
26-158. Ishmael Family—Six Generations of a Degenerate Family.
26-159. Roosevelt Family—Pedigree Chart of a Superior Family.
26-292. Dr. and Mrs. Hugo de Vries.
15-330. Fused Finger Joints.
17-393. Short Legs—Inherited Achondroplasia.
20-75. Extra Fingers—Recessive Polydactylism.
20-74. Chart Showing Inheritance of Polydactylism.
20-352. A Folding Man — Hereditary Absence of Clavicles.
22-104. Crooked Arms and Legs—Hereditary Bone Fragility.
23-307. Five Generations of Glaucoma.
23-425. Inherited Doublejointedness in the Thumb.
24-269. Chart Illustrating Sex-Determination.
22-47. Palm Prints of Fraternal Twins.
22-40. Corresponding Fingerprints of 3 Pairs of Identical Twins.
23-369. Identical Twins—Separated 13 Years.
23-2. Identical Twins Reared Apart—Mary and Mabel when Grown.
23-5. Identical Twins Reared Apart—Mary and Mabel as Babies.
23-6. Identical Twins (Mary and Mabel) at Two Years—Reared Apart.
23-8. Palm Prints of an Identical Twin (Mary)
23-9. Palm Prints of an Identical Twin (Mabel)
24-443. Mirror-Imaging in Eye Dominance in Identical Twins.
24-444. Table Classifying Handedness in Twins.
25-140. Table of Physical Characteristics of Identical Twins Reared Apart.
25-141. Identical Twins (Harold and Holden) Reared Apart.
25-293. Retina of Normal and Albino Eyes.
25-332. Abnormal Fingers and Toes — Syndactyly, Camptodactyly, and "Hammer Toe".
25-333. Short Finger (Brachymetapody) due to short metacarpal bone.
25-391. Hereditary Absence of Incisors.
25-446. Inherited Chest Depression—Koilosternia.
25-483. Sex-Linked Toothlessness.
25-338. Fifty-fifty Segregation for Woolly Hair Mutation.
26-143. Extra Toes—Inherited Polydactylism.
10-389. Twin Acrobats—Identical Twins.
10-409. Typical Identical Twins.
20-50. Identical Twins Reared Apart.
20-52. Palm Patterns of Identical Twins.
20-155. City Boy and Country Boy—Identical Twins Reared Apart.
20-160. Enlarged Prints of Left Ring Finger of Identical Twins (C & O).
22-202. The Hilton Siamese Twins.

ANIMAL

16-154. Pelvic Bones of Normal and Rumpless Fowls.
16-352. Leg Bones of "Reptilian Fowl" and of Normal.
19-413. A Fertile Mare Mule.
19-414. "Old Beck" (Mule) and Her Horse-Like Colt.
19-415. Mule-Like Colt of Fertile Mare Mule.
20-112. Recessive Leg Feathering in Fowl.
20-113. "Heel Tufts" in Fowl.
21-323. Heterogeneous Chromosome Structure — Irregular Cell Division in Drosophila Melanogaster.
22-213. Conjoined Twins in Cattle.
22-296. Backcrosses between Hybrid (Zebu × Yak) Female × Zebu Male.
22-298. Zebu Bull Used in Yak Crosses.
22-299. Yak Cow Used in Zebu Crosses.
22-305. A Zebu-Yak Hybrid—4 Years Old.
22-305. Zebu-Yak Hybrid Cow—4 Years Old.
23-449. A "Flightless" Rooster.
24-183. Parallel Color Mutations (Himalayan and Siamese) in Rabbit and Cat.
25-208. Five Generations of Inbreeding—Shorthorn Cattle (chart).
25-266. X-Ray Photos of Normal and "Flightless" Feathers in Fowl.
25-435. Production of Agglutinins in Rabbits (chart).
25-432. Agglutination of Incompatible Bloods.
25-464. "Portraits" of Salivary Gland Chromosome.
25-466. Giant Chromosomes Compared with "Normal".
25-469. Synapsis of Normal and Inverted Chromosomes.
25-470. Synapsis of Deleted and Normal Chromosomes.
25-472. Cyto-Genetic Maps of Salivary Chromosomes. (Insert.)
26-62. Salivary Chromosomes and Gonial Chromosomes Compared.
26-62. Reference Map of the Salivary Chromosomes of Drosophila. (Insert.)
26-182. Structure of Salivary Gland Chromosome.
26-301. Effects of Sex Hormone Injection on Feather Growth.

PLANT

15-204. Capsules of Globe Daturas.
17-372. Awn of Manchuria (Rough) Barley.
17-373. Awn of Lion (Smooth) Barley.
18-128. Adaptive Cupped Tendrils of a Plant.
18-129. Adaptive "Vacuum Cup" Toes of a Toad.
18-382. Leaves of Live Oak × Post Oak Hybrids.
18-383. Leaves of Overcup × Live Oak Hybrids.
19-419. "Fatback" and "Razorback" Honey Locust Pods.
20-24. Haploid and Dwarf Diploid Tomato Plants.
20-25. Haploid and Normal Tomato Plants.
20-26. Haploid and Diploid Tomatoes.
20-206. Flax Hybrid Produced by Artificial Nutrition.
22-368, 370. Plant Patents No. 2 and No. 3.
24-131. Radium-Induced "Ridged" Tomato.
24-133. Chimeral Radium-Treated Tomato Leaves.
24-134. Radium-Induced Virescent-White Tomato Seedlings.
24-368. Gene Mutation Chimera in Delphinium.
26-137. Ears of Maize, Relatives, and Hybrids.
25-448. Fasciated Sugar Beet.

Above are listed ninety Lantern Slides from JOURNAL OF HEREDITY illustrations. These are only a sample of over five thousand charts and photographs which have been published in the twenty-six volumes of the JOURNAL. Order from this list by Number. Slides may be ordered for any illustration appearing in the JOURNAL by giving Volume—Page—and Figure Number. Slides on this list will be supplied within a week of receipt of order. Price 75c each —$8.00 per dozen. Slides are numbered according to volume and page on which they appear. Thus 25-372 is from Volume 25, page 372 of the JOURNAL, 24-300 is from Volume 24, page 300, etc. Suggestions for illustrative material from the JOURNAL suitable for specific lectures will gladly be furnished on request. Address orders and send for list of 250 slides to:

AMERICAN GENETIC ASSOCIATION

Victor Building **Washington, D. C.**